

TRIPHENYL PHOSPHATE

HSDB - Hazardous Substances Data Bank

0.0 ADMINISTRATIVE INFORMATION

Hazardous Substances Data Bank Number: 2536
Last Revision Date: 970423
Review Date: Reviewed by SRP on 6/15/92
Update History:

1 Complete Update on 04/23/97, 1 field added/edited/deleted.
2 Complete Update on 06/24/96, 7 fields added/edited/deleted.
3 Field Update on 06/11/96, 1 field added/edited/deleted.
4 Complete Update on 01/23/96, 1 field added/edited/deleted.
5 Complete Update on 01/24/95, 1 field added/edited/deleted.
6 Complete Update on 12/28/94, 1 field added/edited/deleted.
7 Complete Update on 08/09/94, 1 field added/edited/deleted.
8 Complete Update on 03/25/94, 1 field added/edited/deleted.
9 Complete Update on 01/12/94, 65 fields added/edited/deleted.
10 Field Update on 08/03/93, 1 field added/edited/deleted.
11 Field update on 12/25/92, 1 field added/edited/deleted.
12 Field Update on 08/26/92, 1 field added/edited/deleted.
13 Complete Update on 01/28/92, 1 field added/edited/deleted.
14 Field update on 01/28/91, 1 field added/edited/deleted.
15 Complete Update on 10/10/90, 1 field added/edited/deleted.
16 Complete Update on 04/16/90, 1 field added/edited/deleted.
17 Complete Update on 03/06/90, 1 field added/edited/deleted.
18 Field update on 03/06/90, 1 field added/edited/deleted.
19 Complete Update on 12/19/89, 1 field added/edited/deleted.
20 Complete Update on 10/31/89, 5 fields added/edited/deleted.
21 Complete Update on 06/04/85

1.0 SUBSTANCE IDENTIFICATION

Name of Substance: TRIPHENYL PHOSPHATE
CAS Registry Number: 115-86-6
Related HSDB Records: No additional data
Synonyms:

1 TPP [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry ofToxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS fi*
2 TRIPHENYLPHOSPHATE [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 125*
3 CELLUFLEX TPP [Peer Reviewed]
4 DISFLAMOLL TP [Peer Reviewed]
5 PHENYL PHOSPHATE ((PHO)3PO) [Peer Reviewed]
6 PHOSFLEX TPP [Peer Reviewed]
7 PHOSPHORIC ACID, TRIPHENYL ESTER [Peer Reviewed]
8 TP [Peer Reviewed]
9 TRIPHENOXYPHOSPHINE OXIDE [Peer Reviewed]

Molecular Formula: C18-H15-O4-P [Peer Reviewed]

Molecular Formula: C18-H15-O4-P [Peer Reviewed]
Wiswesser Line Notation: ROPO&OR&OR [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry ofToxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS fi*
RTECS Number: NIOSH/TC8400000

2.0 MANUFACTURING/USE INFORMATION

Methods of Manufacturing:

1 ... FROM PHOSPHORUS PENTOXIDE AND PHENOL ... BY REACTION OF TRIETHYL PHOSPHITE WITH CHLORAMINE-T. [Peer Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1533*

2 Prepared by reacting phosphorus pentoxide and phenol and by reaction of triethyl phosphate and chloramine-T. On a larger scale phosphorus oxychloride and phenol are reacted in an esterification tank with heating. The hydrogen chloride formed is trapped and condensed, while the crude triphenyl phosphate runs into a large tank where it is purified. [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 488*

Manufacturers:

1 FMC Corporation, Hq, 200 E Randolph Dr, Chicago, IL 60601, (312) 861-6000; Chemical Products Group, 2000 Market St, Philadelphia, PA 19103; Production site: Nitro, WV 25143 [Peer Reviewed] *SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. 883*

2 Monsanto Co, Hq, 800 N Lindbergh Blvd, St Louis, MO 63167, (314) 694-1000; Monsanto Chemical Co (address same as Hq); Production site: Sauget, IL 62201 [Peer Reviewed] *SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. 883*

Other Manufacturing Information:

1 THOUGH IT EASILY SUPERCOOLS, TRIPHENYLPHOSPHATE CAN CRYSTALLIZE WHEN PRESENT IN FILMS, IMPARTING A MILKY APPEARANCE TO THEM. ANY EXCESS OF TRIPHENYLPHOSPHATE SHOULD ... BE AVOIDED; THE ADDITION OF PHTHALATES PREVENTS CRYSTALLIZATION. [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 125*

2 ORGANOLEPTIC INVESTIGATIONS SHOWED THAT MODEL MEDIA (DISTILLED WATER, 1% ACETIC ACID, 0.3% LACTIC ACID) CONTAINING ETROL (CELLULOSE ESTERS USED IN THERMOPLASTIC COMPOSITIONS) PLASTICIZED WITH TRIPHENYL PHOSPHATE ACQUIRED THE ACRID TASTE & ODOR OF THE PLASTICIZER & THEREFORE TRIPHENYL PHOSPHATE IS NOT RECOMMENDED FOR CONTACT WITH FOOD PRODUCTS. [Peer Reviewed] *ANTONYUK OK; GIG SANIT (8): 98 (1974)*

3 ... LESSENS THE INFLAMMABILITY OF PLASTICS IN WHICH IT IS INCORPORATED, PARTICULARLY CELLULOSE ACETATE FILMS. IT IS AN EXCELLENT PLASTICIZER FOR ETHYLCELLULOSE & A VERY GOOD FIREPROOFING AGENT FOR VINYL & ACRYLIC MATERIALS. [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 124*

Major Uses:

1 NONCOMBUSTIBLE SUBSTITUTE FOR CAMPHOR IN CELLULOID; IMPREGNATING ROOFING PAPER; RENDERING ACETYLCELLULOSE, NITROCELLULOSE, AIRPLANE "DOPE" STABLE & FIREPROOF; PLASTICIZER IN LACQUERS & VARNISHES [Peer Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and*

Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1533*

2 PLASTICIZER FOR HOT-MELT ADHESIVES [Peer Reviewed] *SRI*

3 IMPREGNATING UPHOLSTERY [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

4 Fire retarding agent, plasticizer for cellulose acetate and nitrocellulose [Peer Reviewed] *Sax. N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

U.S. Production:

(1975) GREATER THAN 9.08X10+5 GRAMS [Peer Reviewed] *SRI*

3.0 CHEMICAL AND PHYSICAL PROPERTIES

Color/Form:

1 CRYSTALS FROM ABSOLUTE ALCOHOL-LIGROIN, PRISMS FROM ALCOHOL, NEEDLES FROM ETHER-LIGROIN [Peer Reviewed] *Lide, D.R. (ed). CRC Handbook of Chemistry and Physics. 72nd ed. Boca Raton, FL: CRC Press, 1991-1992.,p. 3-398*

2 COLORLESS CRYSTALLINE POWDER [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

3 WHITE PLATELETS [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 124*

4 Colorless, crystalline powder. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

Odor:

1 Odorless [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

2 CHARACTERISTIC ODOR RESEMBLING PHENOL @ ROOM TEMP [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

3 Phenol-like odor. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

Boiling Point: 245 DEG C @ 11 MM HG [Peer Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1533*
Melting Point: 49-50 DEG C [Peer Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1533*
Molecular Weight: 326.28 [Peer Reviewed]
Density/Specific Gravity: 1.2055 @ 50 DEG C/4 DEG C [Peer Reviewed] *Lide, D.R. (ed). CRC Handbook of Chemistry and Physics. 72nd ed. Boca Raton, FL: CRC Press, 1991-1992.,p. 3-398*
Solubilities:

1 0.002% SOL IN WATER @ 54 DEG C; PRACTICALLY INSOL IN PETROL [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 124*

2 SOL IN BENZENE, CHLOROFORM, ETHER, ACETONE; MODERATELY SOL IN ALCOHOL [Peer Reviewed] *Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1533*

3 VERY SOL IN CARBON TETRACHLORIDE [Peer Reviewed] *Weast, R.C. (ed.). Handbook of Chemistry and Physics. 60th ed. Boca Raton, Florida: CRC Press Inc., 1979.,p. C-435*

3 *of Chemistry and Physics. 60th ed. Boca Raton, Florida: CRC Press Inc., 1979.,p. C-435*

4 SOL IN MOST LACQUERS, SOLVENTS, THINNERS, OILS [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

Spectral Properties:

1 SADTLER REFERENCE NUMBER: 9066 (IR, PRISM) [Peer Reviewed] *Weast, R.C. (ed.). Handbook of Chemistry and Physics. 60th ed. Boca Raton, Florida: CRC Press Inc., 1979.,p. C-435*

2 INDEX OF REFRACTION: 1.550 @ 60 DEG C [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

3 Intense mass spectral peaks: 65 m/z (100%), 77 m/z (83%), 326 m/z (67%), 325 m/z (62%) [Peer Reviewed] *Hites, R.A. Handbook of Mass Spectra of Environmental Contaminants. Boca Raton, FL: CRC Press Inc., 1985. 291*

4 IR: 8528 (Sadtler Research Laboratories IR Grating Collection) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 109*

5 UV: 3-682 (Organic Electronic Spectral Data, Phillips et al, John Wiley & Sons, New York) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 109*

6 NMR: 10231 (Sadtler Research Laboratories Spectral Collection) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 109*

7 MASS: 237 (Aldermaston, Eight Peak Index of Mass Spectra, UK) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 109*

8 IR: 2:1038E (Aldrich Library of Infrared Spectra, Aldrich Chemical Co, Milwaukee, WI) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 401*

9 NMR: 10:81C (Aldrich Library of Mass Spectra, Aldrich Chemical Co, Milwaukee, WI) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 401*

10 MASS: 2509 (National Bureau of Standards EPA-NIH Mass Spectra Data Base, NSRDS-NBS-63) [Peer Reviewed] *Weast, R.C. and M.J. Astle. CRC Handbook of Data on Organic Compounds. Volumes I and II. Boca Raton, FL: CRC Press Inc. 1985.,p. V2 401*

Vapor Density: 1.19 (AIR= 1) [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2363*

Vapor Pressure: 1 MM HG @ 193.5 DEG C [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2363*

Other Chemical/Physical Properties:

1 13.32 MG/CU M= APPROX 1 PPM [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.) Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2363*

2 WT/GAL= 10.5 LB [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

3 The Henry's Law constant for triphenyl phosphate can be estimated to be approximately 5.65X10-8 atm cu-m/mole at 25 deg C(SRC). The Henry's Law constant for triphenyl phosphate can also be estimated to be 3.98X10-8 atm-cu m/mole at 25 deg C using a chemical structure estimation method(4,SRC). [Peer Reviewed] *(4) Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991)*

4.0 SAFETY AND HANDLING

FLAMMABLE PROPERTIES

Fire Potential:

FIRE HAZARD: SLIGHT, WHEN EXPOSED TO HEAT OR FLAME. SPONTANEOUS HEATING: NO. [Peer Reviewed] *Sax, N.I. Dangerous Properties of Industrial Materials. 6th ed. New York, NY: Van Nostrand Reinhold, 1984. 2684*

NFPA Hazard Classification:

1 Health: 2. 2= Materials that, on intense or continued (but not chronic) exposure, could cause temporary incapacitation or possible residual injury, including those requiring the use of respiratory protective equipment that has an independent air supply. These materials are hazardous to health, but areas may be entered freely if personnel are provided with full-face mask self-contained breathing apparatus that provides complete eye protection. [Peer Reviewed] *National Fire Protection Guide. Fire Protection Guide on Hazardous Materials. 10 th ed. Quincy, MA: National Fire Protection Association, 1991.,p. 325M-91*

2 Flammability: 1. 1= Includes materials that must be preheated before ignition will occur, such as Class IIIB combustible liquids and solids and semi-solids whose flash point exceeds 200 deg F (93.4 deg C), as well as most ordinary combustible materials. Water may cause frothing if it sinks below the surface of the burning liquid and turns to steam. However, a water fog that is gently applied to the surface of the liquid will cause a frothing which will extinguish the fire. [Peer Reviewed] *National Fire Protection Guide. Fire Protection Guide on Hazardous Materials. 10 th ed. Quincy, MA: National Fire Protection Association, 1991.,p. 325M-91*

3 Reactivity: 0. 0= Includes materials that are normally stable, even under fire exposure conditions, and that do not react with water. Normal fire fighting procedures may be used. [Peer Reviewed] *National Fire Protection Guide. Fire Protection Guide on Hazardous Materials. 10 th ed. Quincy, MA: National Fire Protection Association, 1991.,p. 325M-91*

Flammable Limits:

Noncombustible [Peer Reviewed] *ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 547*

Flash Point:

428 DEG F (CLOSED CUP) [Peer Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS(NIOSH) Publication No. 90-117. Washington, DC: U.S. Government Printing Office, June 1990 220*

FIRE FIGHTING INFORMATION

Fire Fighting Procedures:

CARBON DIOXIDE, DRY CHEMICAL ... [Peer Reviewed] *Sax, N.I. Dangerous Properties of Industrial Materials. 6th ed. New York, NY: Van Nostrand Reinhold, 1984. 2684*

Toxic Combustion Products:

Hazardous decomposition products: Toxic gases and vapors (such as phosphoric acid fume and

carbon monoxide) may be released in a fire involving triphenyl phosphate. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

HAZARDOUS REACTIONS

Decomposition:

1 Hazardous decomposition products: Toxic gases and vapors (such as phosphoric acid fume and carbon monoxide) may be released in a fire involving triphenyl phosphate. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

2 HEATED TO DECOMPOSITION, CAN EMIT HIGHLY TOXIC FUMES OF /PHOSPHORUS OXIDES./ [Peer Reviewed] *Sax, N.I. Dangerous Properties of Industrial Materials. 6th ed. New York, NY: Van Nostrand Reinhold, 1984. 2684*

PREVENTIVE MEASURES

Protective Equipment and Clothing:

1 Wear rubber gloves, self-contained breathing apparatus and coveralls. [Peer Reviewed] *ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 548*

2 Respiratory protection (supplied-air respirator with full facepiece or self-contained breathing apparatus) should be available where these compounds are manufactured or used and should be worn in case of emergency and overexposure. /Phosphorus compounds/ [Peer Reviewed] *International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 1684*

3 Recommendations for respirator selection. Max concn for use: 15 mg/cu m: Respirator Class: Any dust respirator. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

4 Recommendations for respirator selection. Max concn for use: 30 mg/cu m: Respirator Classes: Any dust respirator except single-use and quarter-mask respirators. Any supplied-air respirator. Any self-contained breathing apparatus. Any air-purifying respirator with a high-efficiency particulate filter (if an independent code); or a high-efficiency particulate filter. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

5 Recommendations for respirator selection. Max concn for use: 75 mg/cu m: Respirator Classes: Any powered, air-purifying respirator with a dust and mist filter. Any supplied-air respirator operated in a continuous flow mode. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

6 Recommendations for respirator selection. Max concn for use: 150 mg/cu m: Respirator Classes: Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter. Any powered, air-purifying respirator with a tight-fitting facepiece and a high-efficiency particulate filter. Any supplied-air respirator that has a tight-fitting facepiece and is operated in a continuous-flow mode. Any self-contained breathing apparatus with a full facepiece. Any supplied-air respirator with a full facepiece. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

7 Recommendations for respirator selection. Max concn for use: 1000 mg/cu m: Respirator Class: Any supplied-air respirator operated in a pressure-demand or other positive-pressure

Class: Any supplied-air respirator operated in a pressure-demand or other positive-pressure mode. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

8 Recommendations for respirator selection. Condition: Emergency or planned entry into unknown concn or IDLH conditions: Respirator Classes: Any self-contained breathing apparatus that has a full facepiece and is operated in a pressure-demand or other positive pressure mode. Any supplied-air respirator with a full face piece and operated in pressure-demand or other positive pressure mode in combination with an auxiliary self-contained breathing apparatus operated in pressure-demand or other positive pressure mode. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

9 Recommendations for respirator selection. Condition: Escape from suddenly occurring respiratory hazards: Respirator Classes: Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter. Any appropriate escape-type, self-contained breathing apparatus. [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

Other Preventative Measures:

... HANDLE ... WITH CAUTION, PREVENT CONTACT WITH SKIN, & KEEP AIR CONCN BELOW RECOMMENDED VALUES. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2374*

OTHER SAFETY AND HANDLING

Cleanup Methods:

If triphenyl phosphate is spilled ... : ventilate areas of spill. For small quantities, sweep onto paper or other suitable material, place in appropriate container and burn in a safe place (such as a fume hood). Large quantities may be retained; however, if this is not practical, dissolve in a flammable solvent (such as alcohol) and atomize in a suitable combustion chamber equipped with an appropriate effluent gas cleaning device. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

Disposal Methods:

1 Group I Containers: Combustible containers from organic or metallo-organic pesticides (except organic mercury, lead, cadmium, or arsenic compounds) should be disposed of in pesticide incinerators or in specified landfill sites. /Organic or metallo-organic pesticides/ [Peer Reviewed] *40 CFR 165.9(a) (7/1/91)*

2 Group II Containers: Non-combustible containers from organic or metallo-organic pesticides (except organic mercury, lead, cadmium, or arsenic compounds) must first be triple-rinsed. Containers that are in good condition may be returned to the manufacturer or formulator of the pesticide product, or to a drum reconditioner for reuse with the same type of pesticide product, if such reuse is legal under Department of Transportation regulations (eg 49 CFR 173.28). Containers that are not to be reused should be punctured ... and transported to a scrap metal facility for recycling, disposal or burial in adesignated landfill. /Organic or metallo-organic pesticides/ [Peer Reviewed] *40 CFR 165.9(b) (7/1/91)*

3 1. BY MAKING PACKAGES OF TRIPHENYL PHOSPHATE IN PAPER OR OTHER FLAMMABLE MATERIAL & BURNING IN A SUITABLE COMBUSTION CHAMBER ... WITH AN APPROPRIATE EFFLUENT GAS CLEANING DEVICE. 2. BY DISSOLVING ... IN A

AN APPROPRIATE EFFLUENT GAS CLEANING DEVICE. 2. BY DISSOLVING ... IN A FLAMMABLE SOLVENT ... & ATOMIZING IN A SUITABLE COMBUSTION CHAMBER ... WITH AN ... EFFLUENT GAS CLEANING DEVICE. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

5.0 TOXICITY/BIOMEDICAL EFFECTS

SUMMARY

Medical Surveillance:

Employees should be screened for history of neuromuscular disorders which might place the employee at great risk from triphenyl phosphate exposure. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

TOXICITY EXCERPTS

Human Toxicity Excerpts:

1 /IN A/ ... TRIPHENYL & TRICRESYL PHOSPHATE MANUFACTURING PLANT WITH AIR CONCN MEASUREMENTS RANGING FROM 0.27 TO 3.4 MG/CU M ... NO CORRELATION /WAS FOUND/ BETWEEN RESULTS FROM CHOLINESTERASE DETERMINATIONS & MINOR GI OR NEUROMUSCULAR SYMPTOMS & DEGREE & DURATION OF EXPOSURE. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2369*

2 FOURTEEN EMPLOYEES WERE EXPOSED TO ... VAPOR, MIST OR DUST OVER A PERIOD OF 8-10 YR. PARTICLE SIZES OF DUSTS MEASURED LESS THAN 1 MICRON IN DIAMETER FOR 90% & AVG AIR CONCN WAS ... 3.5 MG/CU M WITH OCCASIONAL EXCURSIONS TO 40 MG/CU M. RESULTS SHOWED NO SIGNS OF ILLNESS, BUT A SLIGHT ... REDUCTION IN RBC CHOLINESTERASE ACTIVITY. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2369*

3 LARGE QUANTITIES OF TRIPHENYL PHOSPHATE INHIBIT HUMAN CHOLINESTERASE IN VITRO & IN VIVO; HOWEVER, IT IS NOT CONSIDERED A POTENT ANTICHOLINESTERASE. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2380*

4 ONE CASE OF /SKIN/ SENSITIZATION HAS BEEN RECORDED. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2381*

5 A case of polyneuropathy in a 48 yr old mechanic with a 2 yr history of heavy skin exposure to hydraulic fluids containing isopropylated triphenyl phosphate is described. The hydraulic fluids contained about 0.5% isopropylated triphenyl phosphate. He had weakness of the hands and absence of deep tendon reflexes. Nerve conduction velocities were normal but there was a marked reduction in motor unit potentials. Three years after transfer to another department his symptoms have improved slightly. A cross-sectionsl examination of 8 other men exposed to hydraulic fluids (although less heavily than the index case) and 8 controls showed differences in electromyography (EMG) but not in nerve conduction velocities. Four of the exposed men showed a reduced number of motor unit potentials and some single potentials of increased duration and amplitude, but none of the controls had such changes.

potentials of increased duration and amplitude, but none of the controls had such changes. /Isopropylated triphenyl phosphate/ [Peer Reviewed] *Jarvholm B et al; Am J Ind Med 9: 561-66 (1986)*

6 TOXIC BY INHALATION. [Peer Reviewed] *Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1192*

7 IN VITRO STUDIES INDICATE THAT CHOLINESTERASE INHIBITION BY TRIPHENYL PHOSPHATE IS GREATER IN HUMAN BLOOD THAN IN RODENT BLOOD. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

8 In workers engaged in the manufacture of aryl phosphates (including triphenyl phosphates and up to 20% triorthocresyl phosphate) and exposed to concentrations of aryl phosphates of 0.2 to 3.4 mg/cu m, there was some inhibition of plasma cholinesterase but no correlation of this effect with degree of exposure or minor gastrointestinal or neuromuscular symptoms. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

9 An allergic reaction in a 67 year old woman to spectacle frames containing triphenyl phosphate was reported. Patch tests with analytical grade triphenyl phosphate in that individual indicated a reaction at concentrations as low as 0.05%. [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 491*

10 Causes a delayed peripheral neuritis involving motor neurons, resulting in a flaccid paralysis, particularly of the distal muscles. No sensory disturbances. [Peer Reviewed] *Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-302*

11 Triaryl phosphates ... include mixed isomers of triphenyl phosphate, tricresyl phosphate, trixylenyl phosphate, & ... /others/. Of these, the ortho isomer of tricresyl phosphate ... is by far the most toxic. /Triaryl phosphates/ [Peer Reviewed] *Clarke, M. L., D. G. Harvey and D. J. Humphreys. Veterinary Toxicology. 2nd ed. London: Bailliere Tindall, 1981. 186*

12 ... NO INDUSTRIAL HUMAN POISONINGS HAVE BEEN REPORTED, EXCEPT FOR ONE /SRP: OCCURRENCE/ ... IN GREAT BRITAIN WHERE TRIPHENYL PHOSPHATE WAS SUSPECTED TO HAVE PLAYED A PART IN EFFECTS BESIDE THE TRITOLYL PHOSPHATE. NO ADVERSE ... EFFECTS ... FOUND IN MEN EXPOSED TO ... VAPOR MIST & DUST FOR 10 YR @ A CONCN OF 3.5 MG/CU M. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2380*

13 NUMEROUS MEDICAL OBSERVATIONS HAVE BEEN MADE ON WORKMEN EMPLOYED FOR SEVERAL (2-10) YR IN THE FACTORY WHERE TRIPHENYL PHOSPHATE WAS MADE. NO ABNORMAL SYMPTOMS APPEAR TO HAVE BEEN FOUND, & IN PARTICULAR NO CASE OF NEUROLOGICAL TROUBLE. [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 127*

Non-Human Toxicity Excerpts:

1 ... PRODUCE/S/ FLACCID PARALYSIS IN ... DOG/S/ ... . [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2372*

2 CHOLINESTERASE IN PLASMA IS INHIBITED TO 50% BY ORAL OR IP ADMIN OF 0.2 G/KG TO MICE & 1 G/KG TO WHITE LEGHORN COCKERELS. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

3 ... SC ADMIN OF 0.2 G/KG OF TRIPHENYL PHOSPHATE CAUSES PARALYSIS IN CATS, & ... DOSES BETWEEN 0.3-1.0 G/KG MIGHT BE FATAL. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit*

Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613

4 ... CAUSED GENERALIZED DELAYED ILLNESS & PARALYSIS IN CATS & PRIMATES. THE OBSERVED DEMYELINATION OF SPINAL CORD RESEMBLED THAT OBTAINED WITH TRI-O-CRESYL PHOSPHATE. NEUROMUSCULAR SIGNS WERE OBSERVED ABOVE 0.2 G/KG IN THE CAT. [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2381*

5 ... ORALLY /ADMIN/ FOR 3 MONTHS TO RATS IN DOSES OF ONE HALF OF LD50 (1.8 G/KG) & ONE TENTH OF LD50 (0.38 G/KG) ... CAUSED NO DEATH/S/. /IT WAS/ ... CONCLUDED FROM THE NORMAL GROWTH & CHOLINESTERASE ACTIVITY THAT THESE DOSES HAVE NO CUMULATIVE TOXIC EFFECTS. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

6 ... RATS /FED/ FOR 35 DAYS WITH A DIET CONTAINING EITHER 0.1% OR 0.5% OF TPP ... OBSERVED SLIGHT DEPRESSION IN GROWTH RATES & INCREASED LIVER WEIGHTS IN RATS FED THE 0.5% DIET. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

7 1 PPM ... IN WATER CAUSED DEATH OF ALL SIX EXPOSED GOLDFISH WITHIN 8 HR. AT CONCN OF 3 PPM OR 5 PPM, ALL FISH DIED WITHIN 5 HR & 1 HR, RESPECTIVELY /HISTOLOGICAL CHANGES SIMILAR TO MICE & RAT TPP POISONING/: CONGESTION, DEGENERATION, & HEMORRHAGE FROM SMALLER BLOOD VESSELS, PRONOUNCED MOSTLY IN LIVER, KIDNEY, & BRAIN. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

8 APPLICATION OF TRIPHENYL PHOSPHATE ON SKIN OF RATS AS WELL AS APPLICATION OF TRIPHENYL PHOSPHATE IN ETHANOL SOLN ON SKIN OF MICE CAUSED NO SKIN IRRITATION. ... CONCLUDED THAT, SINCE CHOLINESTERASE IS NOT INHIBITED AFTER APPLICATION ... ON SKIN, THERE WAS NO DERMAL ABSORPTION. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

9 ... IN MONKEYS & CATS /TRIPHENYL PHOSPHATE/ ... CAUSED /SRP: POLYNEUROPATHY/ ... RESEMBLING THAT PRODUCED BY TRITOLYLPHOSPHATE ... RABBITS & CHICKENS WERE RESISTANT. [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 125*

10 ... REPEATED INGESTION BY RABBITS OF DOSES OF TRIPHENYL PHOSPHATE VARYING BETWEEN 0.10 & 1 G/KG BODY WEIGHT GIVES RISE TO KIDNEY DAMAGE, WHICH, HOWEVER, QUICKLY CLEARS UP ... . IP INJECTIONS OF 0.1-0.2 G/KG DO NOT HAVE ANY TOXIC EFFECT, THOUGH SC INJECTIONS OF 1 G/KG WERE FATAL ... . [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 337*

11 WISTAR STRAIN RATS WERE PUT ON DIETS CONTAINING 5 MG, 10 MG, & 100 MG /50, 100, & 1000 UG/RAT/DAY FOR 6 MO/ ... NONE OF THE 3 DOSES INTERFERED WITH GROWTH /AS COMPARED WITH CONTROLS/ ... IMPREGNATION, GESTATION, PARTURITION, & NURSING ARE NORMAL IN THE SERIES (SMALL DOSE) FOR WHICH ... INVESTIGATION OF REPRODUCTIVE FUNCTIONS WERE CARRIED OUT. [Peer Reviewed] *Lefaux, R. Practical Toxicology of Plastics. Cleveland: CRC Press Inc., 1968. 337*

12 REPORTS ON THE DELAYED NEUROTOXICITY OF TRIPHENYL PHOSPHATE IN THE CAT WERE REEVALUATED IN LIGHT OF DISCOVERIES OF IMPURITIES IN OLD

12 CAT WERE REEVALUATED IN LIGHT OF DISCOVERIES OF IMPURITIES IN OLD SAMPLES & THE NONNEUROTOXICITY OF TRIPHENYL PHOSPHATE IN HENS. IN A LIMITED STUDY USING 99.99% PURE SYNTHETIC TRIPHENYL PHOSPHATE, CATS GIVEN SC DOSES OF 0.4 G/KG DID NOT BECOME ATAXIC; HIGHER DOSES CAUSED PROSTRATION SOME TIME AFTER DOSING BUT HISTOL EXAM DID NOT REVEAL ANY EVIDENCE OF AXON DEGENERATION OR DEMYELINATION IN THE SPINAL CORD. THERE WAS NO EVIDENCE THAT PURE TRIPHENYL PHOSPHATE CAUSED DELAYED NEUROTOXIC EFFECTS OF THE TYPE PRODUCED BY TRI-O-CRESYL PHOSPHATE. [Peer Reviewed] *WILLS JH ET AL; TOXICOL LETT 4 (1): 21 (1979)*

13 CLINICAL CASES OF NEUROLOGICAL TOXICITY IN CATTLE ASSOCIATED WITH PROBABLE EXPOSURE TO HYDRAULIC FLUIDS & SYNTHETIC LUBRICANTS CONTAINING TRIARYL PHOSPHATES (INCLUDING TRIPHENYL PHOSPHATE) PROMPTED EXPERIMENTAL ORAL DOSING OF CATTLE, GOATS, & RABBITS. DYSPNEA, TYMPANITES, INCOORDINATION, PARALYSIS, & DEATH WERE CHARACTERISTIC SIGNS OF TOXICITY. CHOLINESTERASE LEVELS IN RBC WERE ALSO DECREASED. [Peer Reviewed] *DOLLAHITE JW, PIERCE KR; AM J VET RES 30 (AUG): 1461 (1969)*

14 CLINICAL CASES OF NEUROLOGICAL TOXICITY IN CATTLE ASSOCIATED WITH PROBABLE EXPOSURE TO HYDRAULIC FLUIDS & SYNTHETIC LUBRICANTS CONTAINING TRIARYL PHOSPHATES (INCLUDING TRIPHENYL PHOSPHATE) PROMPTED EXPERIMENTAL ORAL DOSING OF CHICKENS & RATS. DYSPNEA, TYMPANITES, INCOORDINATION, PARALYSIS, & DEATH WERE CHARACTERISTIC SIGNS OF TOXICITY IN CHICKENS WITH NO TOXICITY NOTED IN RATS. [Peer Reviewed] *DOLLAHITE JW, PIERCE KR; AM J VET RES 30 (AUG): 1461 (1969)*

15 THE SYMPATHETIC GANGLIA FROM CHICK EMBRYO WERE USED AS A MODEL TO STUDY THE RELATIVE NEUROTOXIC PROPERTIES OF BOTH ESTERASE INHIBITORY & NON-INHIBITORY ORGANOPHOSPHATES. TEN DAY OLD GANGLIONIC EXPLANTS WERE CULTURED FOR 72 HR INCUBATION PERIODS IN A SUITABLE MEDIUM CONTAINING VARIABLE CONCN OF ORGANOPHOSPHORUS CMPD. MICROSLIDES WERE EXAM FOR ABNORMALITIES IN GROWTH PATTERN, CELLULAR CONSTITUENTS, DENSITIES OF NERVE FIBERS, GLIAL CELLS, & NEURONS. SOME OR NO EFFECTS AT 10 MILLIMOLAR WERE EVIDENT FOR TRIPHENYL PHOSPHATE. [Peer Reviewed] *OBERSTEINER EJ, SHARMA RP; FED PROC (FED AM SOC EXP BIOL) 35 (3): 504 (1976)*

16 Triphenyl phosphate was tested for mutagenicity in the Salmonella/microsome preincubation assay using a protocol approved by the National Toxicology Program. Triphenyl phosphate was tested at doses of 0, 100, 333, 1000, 3333, and 10,000 ug/plate in four Salmonella typhimurium strains (TA98, TA100, TA1535, and TA1537) in the presence and absence of Aroclor-induced rat or hamster liver S9. Triphenyl phosphate was negative in these tests and the highest ineffective dose level tested (not causing the formation of a precipitate) in any Salmonella tester strain was 1000 ug/plate. [Peer Reviewed] *Zeiger A et al; Environ Mutagen 9: 1-110 (1987)*

17 Triphenyl phosphate is a neurotoxin in animals. When injected in cats it caused delayed paralysis. Two of 6 cats given a single ip injection of triphenyl phosphate at 0.1 to 0.4 g/kg developed paralysis after 16 to 18 days. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

18 Vertebral damage and spinal curvature were observed in rainbow trout exposed to tripheny phosphate for 96 hr. [Peer Reviewed] *Murty, A.S. Toxicity of Pesticides to Fish. Volumes I, II. Boca Raton, FL: CRC Press Inc., 1986. 57*

19 Experimentally, degeneration of motor nerve cells and peripheral nerves has been induced in animals by means of triorthocresyl phosphate ... and triphenyl phosphate, and lesions have been observed histologically in the nuclei of the nerves of the extraocular muscles of animals. However, no abnormality of oculomotion seems to have been noted in animals or

patients. [Peer Reviewed] *Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. 943*

20 SRP: Although TPP causes delayed neurotoxicity most likely unrelated to cholinesterase inhibition, it does seem to have more anticholinesterase activity than the alkyl phosphates or cresyl phosphates. [Peer Reviewed]

21 ... A mixture of triaryl phosphates (Cellulube 220) /was fed/ to calves & goats. The animals developed anorexia, diarrhea, tympanitis, dyspnea, incoordination & paralysis, ending in death. No gross post mortem lesions were observed. Microscopically there was demyelination & swelling of the axon cylinders, with vacuolation of the cytoplasm of large neurons of the ventral motor nucleus of the spinal cord. /Triaryl phosphates (cellulube 220)/ [Peer Reviewed] *Clarke, M. L., D. G. Harvey and D. J. Humphreys. Veterinary Toxicology. 2nd ed. London: Bailliere Tindall, 1981. 186*

TOXICITY VALUES

Non-Human Toxicity Values:

1 LD50 MOUSE ORAL 1.32 + OR - 0.28 G/KG [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

2 LD50 RAT ORAL 3.8 + OR - 0.26 G/KG [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

3 LD MONKEY SC 0.5 G/KG [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

4 LD50 Rat Sprague Dawley oral 10.8 g/kg [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 489*

5 LD50 Rat oral 3.8 g/kg (in oil) [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 489*

6 LD50 Mouse oral 1.3 g/kg (in oil) [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 489*

7 LD50 White leghorn chicken oral > 5.0 g/kg [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 489*

8 LD50 Rabbit dermal > 7.9 g/kg [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 490*

Ecotoxicity Values:

1 LC50 Pimephales promelas (fathead minnow) 0.87 mg/l/96 hr (confidence limit 0.81- 0.94 mg/l), flow-through bioassay with measured concentrations, 24.5 deg C, dissolved oxygen 6.4 mg/l, hardness 45.6 mg/l calcium carbonate, alkalinity 43.4 mg/l calcium carbonate, and pH 7.78. [Peer Reviewed] *Geiger D.L., Poirier S.H., Brooke L.T., Call D.J., eds. Acute Toxicities of Organic Chemicals to Fathead Minnows (Pimephales Promelas). Vol. III. Superior,Wisconsin: University of Wisconsin-Superior, 1986. 311*

2 EC50 Pimephales promelas (fathead minnow) 0.51 mg/l/96 hr (confidence limit 0.47- 0.56 mg/l), flow-through bioassay with measured concentrations, 24.5 deg C, dissolved oxygen 6.4 mg/l, hardness 45.6 mg/l calcium carbonate, alkalinity 43.4 mg/l calcium carbonate, and

6.4 mg/l, hardness 45.6 mg/l calcium carbonate, alkalinity 43.4 mg/l calcium carbonate, and pH 7.78. Effect: loss of equilibrium. [Peer Reviewed] *Geiger D.L., Poirier S.H., Brooke L.T., Call D.J., eds. Acute Toxicities of Organic Chemicals to Fathead Minnows (Pimephales Promelas). Vol. III. Superior,Wisconsin: University of Wisconsin-Superior, 1986. 311*

3 LC50 Lepomis Macrochirus 290 ppm/96 hr, static bioassay in fresh water at 23 deg C, mild aeration applied after 24 hr. [Peer Reviewed] *Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 117*

4 LC50 Menidia beryllina 95 ppm/96 hr, static bioassay in synthetic seawater at 23 deg C, mild aeration applied after 24 hr. [Peer Reviewed] *Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 1172*

5 LC50 Rainbow trout 300 ug/l/96 hr /Conditions of bioassay not specified/ [Peer Reviewed] *Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 1172*

Populations at Special Risk:

Triphenyl phosphate causes neurotoxic effects in animals. Persons with preexisting neuromuscular disorders may be at increased risk. [Peer Reviewed] *Mackison, F. W., R. S. Stricoff, and L. J Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.*

PHARMACOKINETICS

Absorption, Distribution and Excretion:

1 ... POORLY ABSORBED THROUGH THE INTACT SKIN ... . [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2381*

2 ... READILY ABSORBED THROUGH GUINEA PIG SKIN ... . [Peer Reviewed] *Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2372*

3 THE BIOCONCENTRATION RATIO OF TRIPHENYL PHOSPHATE WAS LARGE COMPARED WITH THOSE OF OTHER PHOSPHATES IN BOTH SPECIES. TRIPHENYL PHOSPHATE ORIGINALLY PRESENT IN THE WATER WAS ABSORBED & METABOLIZED COMPLETELY BY KILLIFISH WITHIN 72 HR, WHILE GOLDFISH ABSORBED TRIPHENYL PHOSPHATE AT A VERY SLOW RATE, BUT ACCUMULATED TPP FOR A LONGER PERIOD. THUS, THE ABILITY TO METABOLIZE TRIPHENYL PHOSPHATE IS HIGH IN KILLIFISH WHILE IT IS VERY LOW IN GOLDFISH. [Peer Reviewed] *SASAKI K ET AL; BULL ENVIRON CONTAM TOXICOL 27 (6): 775 (1981)*

4 THE RATE OF UPTAKE OF (14)C-RING LABELED TRIPHENYL PHOSPHATE BY RAINBOW TROUT WAS LOWER IN RIVER THAN IN DECHLORINATED CITY WATER. LIVER ACCUMULATED THE HIGHEST RESIDUES OF TRIPHENYL PHOSPHATE COMPARED TO OTHER TISSUES. A BIOACCUMULATION RATIO OF 2590 WAS FOUND, BASED ON EXPERIMENTALLY DETERMINED UPTAKE & CLEARANCE RATES. [Peer Reviewed] *MUIR D CG ET AL; CHEMOSPHERE 9 (9): 525 (1980)*

5 Most organophosphate compounds are ... absorbed from skin, conjunctiva, GI tract, & lng. /Organophosphate compounds/ [Peer Reviewed] *Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. 1071*

6 APPLICATION OF TRIPHENYL PHOSPHATE ON SKIN OF RATS AS WELL AS APPLICATION OF TRIPHENYL PHOSPHATE IN ETHANOL SOLN ON SKIN OF MICE CAUSED NO SKIN IRRITATION. ... CONCLUDED THAT, SINCE CHOLINESTERASE IS NOT INHIBITED AFTER APPLICATION ... ON SKIN, THERE WAS NO DERMAL ABSORPTION. [Peer Reviewed] *American Conference of Governmental Industrial*

ABSORPTION. [Peer Reviewed] *American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 613*

Metabolism/Metabolites:

1 STUDY DEALS WITH METABOLIC RING-HYDROXYLATION OF TRIPHENYL PHOSPHATE IN HOUSEFLIES TO GIVE A QUINOL PHOSPHATE. A TOTAL OF 530 MG TRIPHENYL PHOSPHATE WAS APPLIED TO ABOUT 27,000 HOUSEFLIES. METABOLITES WERE EXTRACTED AFTER 24 HR. IN VIVO TRANSFORMATION OF TRIPHENYL PHOSPHATE INTO DIPHENYL P-HYDROXYPHENOL PHOSPHATE WAS CONFIRMED. [Peer Reviewed] *ETO M ET AL; BOCHU-KAGAKU 40 (3): 106 (1975)*

2 Rat liver microsomal enzymes degraded triphenyl phosphate in the presence of thereduced form of nicotinamide-adenine dinucleotide phosphate, but also in the absence of the reduced form of nicotinamide-adenine dinucleotide phosphate. The product of incubation was diphenyl phosphate. It was clear that the reaction was cytochrome p450-linked since the reaction was inhibited by carbon monoxide ... . Goldfish liver microsomes metabolized only about 10% of triphenyl phosphate ... . [Peer Reviewed] *Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990. 489*

Interactions:

A SERIES OF SYMMETRICAL TRISUBSTITUTED PHOSPHORUS ESTERS WAS TESTED AS SYNERGISTS FOR MALATHION & OTHER ORGANOPHOSPHORUS INSECTICIDES AGAINST RESISTANT & NORMAL STRAINS OF CHRYSOMYA PUTORIA & MUSCA DOMESTICA. TRIPHENYL PHOSPHATE SYNERGIZED THE INSECTICIDES MORE EFFECTIVELY AGAINST INSECTICIDE-RESISTANT THAN NORMAL INSECT STRAINS. [Peer Reviewed] *BELL JD, BUSVINE JR; ENTOMOL EXP APPL 10 (2): 263 (1967)*

7.0 ENVIRONMENTAL FATE/EXPOSURE POTENTIAL

SUMMARY

Environmental Fate/Exposure Summary:

Triphenyl phosphate (TPP) may be released to the environment from its production, processing, and use. The major sources of release are volatilization from plastic items where it is used as aplasticizer and leaking from hydraulic fluids. If released to water, triphenyl phosphate will rapidly biodegrade under aerobic conditions (half-life of 4 days or less). However, biodegradation in benthic sediments is unclear. Aqueous hydrolysis will become increasingly important with increasing alkalinity; at pH 9 and25 deg C the half-life is about 3 days. The hydrolysis half-life for triphenyl phosphate should range between 20 and 25 days atpH 7 for the first hydrolysis step converting triphenyl phosphate to diphenyl phosphate. Triphenyl phosphate will partition from the water column to sediment. If released to soil, biodegradation will be the predominant fate process and aqueous hydrolysis may be important in alkaline soils. Triphenyl phosphate will adsorb strongly to soil and leaching will not be important. If released to the atmosphere, vapor-phase triphenyl phosphate is expected to degrade by reaction with photochemically produced hydroxyl radicals (estimated half-life of 1.4 days). Particulate triphenyl phosphate will be removed from the atmosphere via wet and dry deposition. Occupational exposure to triphenyl phosphate will occur via dermal contact and inhalation of vapor. The general population may be exposed to triphenyl phosphate through ingestionof contaminated drinking water and food. (SRC) [Peer Reviewed]

POLLUTION SOURCES

POLLUTION SOURCES

Artificial Sources:

Triphenyl phosphate (TPP) and other aryl phosphates are released during production to soil via land disposal or solid and semi-solid waste(1). Triphenyl phosphate may also be released to the environment through processing and use applications; furthermore, release via use applications are expected to be more than from production(1). Triphenyl phosphate may volatilize from plastic items where it is used as a plasticizer, it may leak from hydraulic fluids, and it may be released from lubricants where it is used as an additive(1-3). Triphenyl phosphate may be released from plastics that are exposed to water in piping, it may leach from landfills, and it may be released in wastewater effluents resulting from production and use(1-2). [Peer Reviewed] *(1) Boethling RS, Cooper JC; Res Rev 94: 49-99 (1985) (2) Muir DCG; 3: 41-66 in The Handbook of Environmental Chemistry Germany: Springer-Verlag Berlin (1984) (3) Kalman DA; Am Ind Hyg Assoc J 47: 270-5 (1986)*

ENVIRONMENTAL FATE

1 TRIARYL PHOSPHATE ESTERS, INCLUDING TRIPHENYL PHOSPHATE, EXHIBIT LOW AQUEOUS SOLUBILITY, HAVE MODERATE POTENTIAL FOR BIOCONCENTRATION, & READILY UNDERGO PRIMARY & ULTIMATE BIODEGRADATION BY NATURALLY OCCURRING MIXED-MICROBIAL POPULATIONS PRESENT IN ACTIVATED SLUDGE & RIVER WATER. [Peer Reviewed] *SAEGER V ET AL; ENVIRON SCI TECHNOL 13 (7): 840 (1979)*

2 THE MOBILITY, PERSISTENCE, BIOCONCENTRATION POTENTIAL, ACUTE & CHRONIC TOXICITIES, & ENVIRONMENTAL CONCN OF PYDRAUL 50E & PYDRAUL 115E & THEIR 3 MAIN COMPONENTS, INCLUDING TRIPHENYL PHOSPHATE, WERE STUDIED. PYDRAULS 50E & 115E PRESENTLY DO NOT APPEAR TO CONSTITUTE AN UNREASONABLE RISK TO THE ENVIRONMENT. TOXICITY TESTS INDICATE THAT THE MARGIN OF SAFETY BETWEEN ENVIRONMENTAL CONCN IN WATER & THE EXPECTED BIOLOGICAL EFFECT CONCN IS GREATER THAN OR EQUAL TO 1 ORDER OF MAGNITUDE FOR COLDWATER FISH, & APPROX 3 ORDERS OF MAGNITUDE FOR WARMWATER SPECIES. BETTER CONTROL OF HYDRAULIC FLUID LEAKAGE WOULD REDUCE ENVIRONMENTAL CONTAMINATION & INCREASE THE MARGIN OF SAFETY. [Peer Reviewed] *MAYER FL ET AL; ASTM SPEC TECH PUBL 737 (AQUAT TOXICOL HAZARD ASSESS) 103 (1981)*

3 TERRESTRIAL FATE: Biodegradation is expected to be the dominant fate process of triphenyl phosphate (TPP) in soil(SRC); screening tests exhibited aerobic half-lives of about 4 days or less in natural waters(1-4). Aqueous hydrolysis is expected to aid in soil degradation especially under alkaline conditions (half-lives of 0.23 to 7.5 days at pH 9.5 to pH 8.2)(1-3). Based on an estimated Koc range of 3,066 to 7,481, TPP should be slightly mobile to immobile in soil(SRC). [Peer Reviewed] *(1) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Testing Mater (1981) (2) Howard PH, Deo PG; Bull Environ Contam Toxicol 22: 337-44 (1979) (3) Boethling RS, Cooper JC; R*

4 AQUATIC FATE: Triphenyl phosphate (TPP) is essentially nonvolatile from water based on a low Henry's Law constant(SRC). Biodegradation is an important removal process in aerobic waters with aerobic half-lives of about 4 days or less(4). However, biodegradation in benthic sediments is unclear(4). Hydrolysis in water will be an important fate process under alkaline conditions with measured half-lives for the first hydrolysis step ranging from 0.23 to 7.5 days at pH 9.5 to pH 8.2, respectively(1,2,4,5). The second order hydrolysis half-life is estimated to be about 296 days at pH 7 and 27 deg C(3,SRC). The hydrolysis half-life for triphenyl phosphate should range between 20 and 25 days at pH 7 for the first hydrolysis step converting triphenyl phosphate to diphenyl phosphate(2,4). Several studies have demonstrated that triphenyl phosphate will partition from the water column to sediment and

demonstrated that triphenyl phosphate will partition from the water column to sediment and suspended solids leaving a higher concn of triphenyl phosphate in the sediment than in the water column(4). [Peer Reviewed] *(1) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Testing Mater (1981) (2) Howard PH, Deo PG; Bull Environ Contam Toxicol 22: 337-44 (1979) (3) Wolfe NL et al; Chemospher*

5 ATMOSPHERIC FATE: Based on an extrapolated vapor pressure of 1.1X10-6 mm Hg at 25 deg C(2) for the super cooled liquid, a vapor pressure of 2.5X10-7 mm Hg at 25 deg C can be estimated for triphenyl phosphate (TPP) after converting to the solid phase(SRC). Based on this vapor pressure value, triphenyl phosphate should exist in the particulate and vapor phases in the ambient atmosphere(3). Vapor phase triphenyl phosphate is degraded in the ambient atmosphere by reaction with photochemically formed hydroxyl radicals; the half-life for this reaction in air can be estimated to be about 1.4 days(3,SRC). Particulate phase triphenyl phosphate may be susceptible to dry deposition and wet deposition (rainfall)(SRC). [Peer Reviewed] *(1) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981) (2) Daubert TE, Danner RP; Physical & Thermodynamic Properties of Pure Chemicals Supplement 1 NY, NY: Hemisphere Pub Corp (1991) (3) Atkinson R; Environ Toxicol Chem 7: 435-42 (1988)*

ENVIRONMENTAL TRANSFORMATIONS

Biodegradation:

1 The ultimate biodegradation of triphenyl phosphate (TPP), initial concn of 22 ppm, was observed to be 82% of theoretical CO2 evolution in a CO2-evolution screening study using acclimated seed (14 day acclimation period) over an incubation period of 27 days(2). The ultimate biodegradation of triphenyl phosphate, initial concn of 18.3 ppm, was observed to be 61.9 and 81.8 % of theoretical CO2 evolution in a CO2-evolution screening study using acclimated seed (14 day acclimation period) over an incubation period of 7 and 28 days, respectively(1). Triphenyl phosphate (concn of 3 to 13 ppm) degraded 84 to 96% over 24 hrs in semicontinuous activated sludge (SCAS) tests(1,2,4) and a half-life of 2 to 4 days was observed in river die-away tests for triphenyl phosphate at an initial concn of 0.05 to 1 ppm(1,2). In 3 freshwater grab sample studies (pH 7.8 to 8.2), triphenyl phosphate exhibited 100% biodegradation after a 2 day lag period (7 to 8 day incubation time)(3). [Peer Reviewed] *(1) Saeger VW et al; Environ Sci Technol 13: 840-4 (1979) (2) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Test Mater (1981) (3) Howard PH, Deo PG; Bull Environ Contam T*

2 Triphenyl phosphate (TPP), initial concn of 0.05 ppm, was degraded 50% based on the disappearance of the parent compound over an incubation time of 50 to 60 days in a grab sample study of freshwater sediment and the major product was observed to be diphenyl phosphate(1). A biodegradation half-life of 2.8 days was estimated for triphenyl phosphate at 10 and 25 deg C based on eleven 14CO2 evolution studies in either pond sediment or river sediment(2). [Peer Reviewed] *(1) Muir DCG et al; ACS Symp Series p. 222 (1981) (2) Muir DCG et al; Toxicol Environ Chem 18: 269-86 (1989)*

Abiotic Degradation:

1 Hydrolysis half-lives of 19 days at pH 7 and 3 days at pH 9 have been determined for triphenyl phosphate (TPP) at 25 deg C(1) where as hydrolysis half-lives of 7.5 and 1.3 days at pH 8.2 and pH 9.5, respectively, have been determined at 21 deg C(2). The hydrolysis half-life for triphenyl phosphate should range between 20 and 25 days at pH 7 for the first hydrolysis step converting triphenyl phosphate to diphenyl phosphate(2,4). Based on a second order alkaline rate constant of 0.27 1/M-sec at 27 deg C(3), a hydrolysis half-life of 296 days at pH 7 and 27 deg C can be estimated for triphenyl phosphate(SRC). Hydrolysis half-lives for 3:1 (vol/vol) dioxane-water mixtures (24 deg C) of 472 days at pH 8.2 and 23 days at pH 9.5 have also been calculated(2) which suggest environmental hydrolysis half-lives of 4.7 days at pH 8.2 and 0.23 days at pH 9.5(4). [Peer Reviewed] *(1) Mayer FL et*

half-lives of 4.7 days at pH 8.2 and 0.23 days at pH 9.5(4). [Peer Reviewed] *(1) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Test Mater (1981) (2) Howard PH, Deo PG; Bull Environ Contam Toxicol 22: 337-44 (1979) (3) Wolfe NL et al; Chemosphere 9*

2 The rate constant for the vapor-phase reaction of triphenyl phosphate (TPP) with photochemically produced hydroxyl radicals has been estimated to be 1.14X10-11 cu cm/molecule-sec at 25 deg C which corresponds to an atmospheric half-life of about 1.4 days at an atmospheric concn of 5X10+5 hydroxyl radicals per cu cm(1,SRC). Photolysis is not expected to be important; triphenyl phosphate contains no chromophores that absorb UV radiation > 290 nm(2,SRC). [Peer Reviewed] *(1) Atkinson R; Environ Toxicol Chem 7: 435-42 (1988) (2) Sadtler Res Lab; Sadtler Standard UV Spectra No. 17459 (1971)*

ENVIRONMENTAL TRANSPORT

Bioconcentration:

1 Rates of uptake by rainbow trout (Salmo gairdneri) in short-term static exposures were higher for triphenyl phosphate (TPP) in dechlorinated city water than in river water, suggesting that sorption to suspended solids or dissolved organic matter reduces the bioavailability of triphenyl phosphate in natural waters(4-5). Bioconcentration factors (BCFs) of 180 to 280 have been measured in rainbow trout exposed to Pydraul 50E (a hydraulic fluid containing 35% triphenyl phosphate) for 90 days in flowing water(1). A BCF range of 132 to 364 was observed for rainbow trout exposed to triphenyl phosphate in flowing water for 90 days(2). BCFs of 573 and 561 have been measured using static tests for radiolabelled TPP in rainbow trout and fathead minnows (Pimephales promelas), respectively(3). A BCF of 2,590 was determined for triphenyl phosphate in rainbow trout based on the bioaccumulation ratio (the rate of the initial uptake rate constant to the initial clearance rate constant); however, this represents the worse-case estimate based on the total 14C content of whole fish after 24 hr exposure(5). [Peer Reviewed] *(1) Lombardo P, Egry IJ; J Assoc Offic Anal Chem 62: 47-51 (1979) (2) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Test Mater (1981) (3) Muir DCG et al; Chemosphere 12:*

2 A bioconcentration factor (BCF) for triphenyl phosphate of 250 was measured in killifish under static conditions and a BCF of 155 was measured in killifish in a flow through system(1). A BCF for triphenyl phosphate of 110 was observed in goldfish under static conditions(2). [Peer Reviewed] *(1) Sasaki K et al; Bull Environ Contam Toxicol 27: 775-82 (1981) (2) Sasaki K et al; Bull Environ Contam Toxicol 28: 752-9 (1982)*

Soil Adsorption/Mobility:

Based upon a measured water solubility of 1.9 mg/L at 25 deg C(1) and a measured log Kow of 4.59(2), the Koc range for triphenyl phosphate (TPP) can be estimated to be 3,066 to 7,481 from regression derived equations(3,SRC). According to a suggested classification scheme(4), triphenyl phosphate should be slightly mobile to immobile in soil and sediment. The partitioning of triphenyl phosphate between marine water and sediment (Kd of 60; organic content not stated)(5), the low water solubility, and high Koc range suggest that adsorption to soil and sediment should be an important fate process for triphenyl phosphate(5,SRC). At 24h, the sediment (1.1% organic carbon) water partition coefficient (Kp) for triphenyl phosphate was 112(6). [Peer Reviewed] *(1) Saeger VW et al; Environ Sci Technol 13: 840-4 (1979) (2) Hansch C, Leo AJ; Medchem Project Issue No. 26 Claremont, CA: Pomona College (1985) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington DC: Amer Chem Soc pp. 4-9,*

Volatilization from Soil/Water:

Based on an extrapolated vapor pressure of 1.1X10-6 mm Hg at 25 deg C(2) for the super cooled

Based on an extrapolated vapor pressure of 1.1X10-6 mm Hg at 25 deg C(2) for the super cooled liquid, a vapor pressure of 2.5X10-7 mm Hg at 25 deg C can be estimated for triphenyl phosphate (TPP) after converting to the solid phase(SRC). Based on this vapor pressure value and a measured water solubility of 1.9 mg/L at 25 deg C(1), the Henry's Law constant for triphenyl phosphate can be estimated to be approximately 5.65X10-8 atm cu-m/mole at 25 deg C(SRC). The Henry's Law constant for triphenyl phosphate can also be estimated to be 3.98X10-8 atm-cu m/mole at 25 deg C using a chemical structure estimation method(4,SRC). According to a suggested classification scheme(3), these Henry's Law constants indicate that triphenyl phosphate is essentially nonvolatile from water. [Peer Reviewed] *(1) Saeger VW et al; Environ Sci Technol 13: 840-4 (1979) (2) Daubert TE, Danner RP; Physical & Thermodynamic Properties of Pure Chemicals Supplement 1 NY, NY: Hemisphere Pub Corp (1991) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Method*

ENVIRONMENTAL CONCENTRATIONS

Water Concentrations:

1 DRINKING WATER: Triphenyl phosphate has been qualitatively identified in a drinking water concentrate taken from New Orleans, LA on January 14, 1976(1). Triphenyl phosphate was detected in Great Lakes municipal drinking water supplies drawn from 22 different locations in 21 Canadian cities at concns ranging from 0.2 to 8.6 ng/L between August and December 1979(2). Triphenyl phosphate was quantitatively detected in drinking water from 6 eastern Ontario water treatment plants during June, September, and October 1978 at concns ranging from 0.2 to 2.6 ng/L at an average concn of 0.9 ng/L(3). Triphenyl phosphate has been detected in 2 carbon chloroform extracts taken from a drinking water plant in New Orleans, LA at concns of 0.12 ug/L and 0.03(4). Triphenyl phosphate was tentatively identified in 4 drinking water samples taken from a water treatment plant in Philadelphia, PA(5). [Peer Reviewed] *(1) Lucas SV et al; GC/MS Analysis of Organics in Drinking Water Concentrates & Advanced Waste Treatment Concentrates Vol. 1 pp. 45, 46, 50, 185, USEPA-600/1-84-020a (1984) (2) Williams DT, LeBel GL; Bull Environ Contam Toxicol 27: 450-7 (1981) (3) LeBel*

2 SURFACE WATER: Triphenyl phosphate (TPP) has been detected at concns of 6, 9, and 11 ng/L in 3 Mississippi river water samples during the summer of 1984(1). Triphenyl phosphate has been detected in 5 of 16 river water samples in Kitakyushu city, Japan at concns ranging from 13 to 31 ng/L(2). Triphenyl phosphate has been detected at a concn of 0.1 in 3 of 3 water samples from upper Saginaw River, MI and concns ranging from 0.6 to 0.7 ug/g in 4 of 4 samples from the Saginaw River at Lake Huron(3). During November of 1977 to May of 1978, triphenyl phosphate was detected at concns of 0.1, 0.1, 0.1 to 0.2, 0.1 to 7.9, 0.1 to 0.4, 0.1 to 0.8, and 0.1 ug/L in water samples taken from the Illinois River in Grafton, IL, the Missouri River at Chesterfield, MO, the Missouri River in Halls Ferry, MO, the Mississippi River in St. Louis, MO, the Mississippi River below St. Louis, MO, the Kanawha River in Winfield, WV, and San Francisco Bay, CA, respectively(3). Triphenyl phosphate was identified in Delaware river water samples taken in August 1976 at a concn range of 0.1 to 0.4 ppb and in March 1977 at a concn range of 0.1 to 0.3 ppb(4). [Peer Reviewed] *(1) DeLeon IR et al; Chemosphere 15: 795-805 (1986) (2) Ishikawa S et al; Water Res 19: 119-25 (1985) (3) Mayer FL et al; pp. 103-23 Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds. Amer Soc Test Mater (1981) (4)*

3 RAIN/SNOW: Triphenyl phosphate has been qualitatively identified in rainwater from Los Angeles, CA during the winter season of 1981 to 1982(1). [Peer Reviewed] *(1) Kawamura K, Kaplan IR; Environ Sci Technol 17: 497-501 (1983)*

Effluents Concentrations:

Triphenyl phosphate has been qualitatively identified in advanced waste treatment concentrates from Pomona, CA and Orange County, CA on September 25, 1974 and February 3, 1976,

respectively(1). Triphenyl phosphate has been qualitatively identified in trench leachate samples from low-level radioactive waste disposal sites in Maxey Flats, KY and West Valley, NY(2). Triphenyl phosphate has been qualitatively identified in emissions from coal and/or refuse combustion(3). Triphenyl phosphate has been detected in wastewater effluents from the Nonferrous Metals industry at a concn of 9 ng/uL extract and the Mechanical Products industry at a concn of 27 ng/uL extract(4). [Peer Reviewed] *(1) Lucas SV et al; GC/MS Analysis of Organics in Drinking Water Concentrates & Advanced Waste Treatment Concentrates Vol. 1 pp. 45, 46, 150, 185, USEPA-600/1-84-020a (1984) (2) Francis AJ et al; Nuclear Tech 50: 158-63 (1980) (3) Junk GA, Ford CS; Chemo*

Sediment/Soil Concentrations:

Triphenyl phosphate has been detected at a concn of 0.01 ug/g in 2 of 3 sediment samples from Waukegan Harbor, IL; it has been detected at a concn of 0.01 in 3 of 3 sediment samples from upper Saginaw River, MI and concns ranging from 1 to 4 ug/g in 3 of 3 sediment samples from the Saginaw River at Lake Huron; it has been detected at concns of 0.1 in 2 of 6 sediment samples taken from the Mississippi River in St. Louis, MO and 0.2 ug/g in 3 of 6 sediment samples taken from the Kanawha River in Winfield, WV during November, 1977 to May, 1978(1). [Peer Reviewed] *(1) Mayer FL et al; pp. 103-23 Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds., Amer Soc Test Mater (1981)*

Food Survey Values:

Triphenyl phosphate has been detected in a composite grain products sample (flours, breads, and cereals); however, this food sample may have been contaminated during analysis through laboratory reagents that contain triphenyl phosphate(1). [Peer Reviewed] *(1) Daft JL; Bull Environ Contam Toxicol 29: 221-7 (1982)*

Fish/Seafood Concentrations:

Triphenyl phosphate (TPP) has been detected at a concn of 0.1 ug/g in 1 of 10 muscle samples in fish collected from the Saginaw River at Lake Huron; triphenyl phosphate has been detected at a concns of 0.5 ug/g in 1 of 3 muscle samples and 0.1 ug/g in 1 of muscle samples in fish collected from the Mississippi River above and below St. Louis, MO, respectively; it has also been detected at concns ranging from 0.1 to 0.6 ug/g in 13 of 27 muscle samples in fish collected from the Kanawha River in Winfield, WV(1). In the above study, different species were collected at each site during November, 1977 to May, 1978; however, the specific species containing triphenyl phosphate were not identified(1). Triphenyl phosphate was found at respective concns of 0.112, 0.6, and 0.15 ppm in 2 carp and 1 goldfish taken in Waukegan Harbor, IL near an industrial outfall which discharges Pydraul 50 E (35% triphenyl phosphate)(2). [Peer Reviewed] *(1) Mayer FL et al; pp. 103-23 in Aquatic Toxicity & Hazard Assessment: 4th Conf. ASTM STP 737. Branson DR, Dickson KL Eds. Amer Soc Test Mater (1981) (2) Lombardo P, Egry IJ; J Assoc Offic Anal Chem 62: 47-51 (1979)*

HUMAN EXPOSURE

Probable Routes of Human Exposure:

1 The general public may be exposed to triphenyl phosphate via contaminated food(2-3) and drinking water(SRC). Workers may be exposed to triphenyl phosphate through inhalation of vapors produced via injection molding of plastic process materials(1) and via dermal contact(SRC). [Peer Reviewed] *(1) Kalman DA; Am Ind Hyg Assoc J 47: 270-5 (1986) (2) Daft JL; Bull Environm Contam Toxicol 29: 221-7 (1982) (3) Gunderson EL; J Assoc Off Anal Chem 71: 1200-9 (1988)*

2 Foods sometimes become contaminated with aryl/alkyl phosphates by diffusion from phosphate treated packaging materials. Food samples also become contaminated during

phosphate treated packaging materials. Food samples also become contaminated during analysis through laboratory reagents that contain these esters, eg, bulk alcohols & organic solvents. /Aryl and alkyl phosphates/ [Peer Reviewed] *Daft JL; Bull Environ Contam Toxicol 29 (2): 221-7 (1982)*

3 NIOSH (NOHS Survey 1972-1974) has statistically estimated that 28,220 workers are potentially exposed to triphenyl phosphate in the USA(1). [Peer Reviewed] *(1) NIOSH; National Occupational Hazard Survey (NOHS) (1974)*

Average Daily Intake:

The average daily intake (AVDI) of triphenyl phosphate in 8 population groups in 1982-1984 was determined according to the FDA's monitoring program for chemical contaminants in the U.S. food supply (Total Diet Study or Market Basket Study). In 6-11 month old infants, the AVDI was 0.3 ng/kg-body weight-per day. In 2 yr old toddlers, the AVDI was 4.4 ng/kg-body weight-per day. In 14-16 year old females, the AVDI was 1.6 ng/kg-body weight-per day. In 14-16 year old males, the AVDI was 1.2 ng/kg-body weight-per day. In 25-30 year old females, the AVDI was 0.8 ng/kg-body weight-per day. In 25-30 year old males, the AVDI was 1.6 ng/kg-body weight-per day. In 60-65 year old females, the AVDI was 0.5 ng/kg-body weight-per day. In 60-65 year old males, the AVDI was 0.5 ng/kg- body weight-per day(1). [Peer Reviewed] *(1) Gunderson EL; J Assoc Off Anal Chem 71. 1200-9 (1988)*

Body Burdens:

During a study of the level of triaryl/alkyl phosphates in human adipose tissue, no conclusions for triphenyl phosphate were drawn because trace levels of triphenyl phosphate were identified in the method blanks; however, triphenyl phosphate was detected in two human adipose tissue samples (13.6 and 11.2 ng/g) at levels significantly above the method blanks (3.3 + or - 4.2 ng/g)(1). [Peer Reviewed] *(1) Lebel GL, Williams DT; Bull Environ Contam Toxicol 37: 41-6 (1986)*

8.0 EXPOSURE STANDARDS AND REGULATIONS

STANDARDS AND REGULATIONS

Immediately Dangerous to Life or Health: 1000 mg/cu m [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

OCCUPATIONAL PERMISSIBLE LEVELS

OSHA Standards:

8 hr Time-Weighted avg: 3 mg/cu m [Peer Reviewed] *29 CFR 1910.1000 (7/1/90)*

NIOSH Recommendations: 10 hr Time-Weighted avg: 3 mg/cu m [QC Reviewed] *NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 322*

Threshold Limit Values:

1 8 hr Time Weighted Avg (TWA) 3 mg/cu m (1986) [QC Reviewed] *American Conference of Governmental Industrial Hygienists. Threshold Limit Values (TLVs) for Chemical Substances and Physical Agents and BiologicalExposure Indices (BEIs) for 1995-1996. Cincinnati, OH: ACGIH, 1995. 35*

2 Excursion Limit Recommendation: Excursions in worker exposure levels may exceed three times the TLV-TWA for no more than a total of 30 min during a work day, and under no

times the TLV-TWA for no more than a total of 30 min during a work day, and under no circumstances should they exceed five times the TLV-TWA, provided that the TLV-TWA is not exceeded. [QC Reviewed] *American Conference of Governmental Industrial Hygienists. Threshold Limit Values (TLVs) for Chemical Substances and Physical Agents and BiologicalExposure Indices (BEIs) for 1995-1996. Cincinnati, OH: ACGIH, 1995. 5*

OTHER STANDARDS AND REGULATIONS

TSCA Requirements:

1 Pursuant to section 8(d) of TSCA, EPA promulgated a model Health and Safety Data Reporting Rule. The section 8(d) model rule requires manufacturers, importers, and processors of listed chemical substances and mixtures to submit to EPA copies and lists of unpublished health and safety studies. Phosphoric acid, triphenyl ester is included on this list. [Peer Reviewed] *40 CFR 716.120 (7/1/90)*

2 Section 8(a) of TSCA requires manufacturers of this chemical substance to report preliminary assessment information concerned with production, use, and exposure to EPA as cited in the preamble of the 51 FR 41329. [Peer Reviewed] *40 CFR 712.30 (7/1/90)*

FDA Requirements:

Triphenylphosphate is an indirect food additive for use only as a component of adhesives. [Peer Reviewed] *21 CFR 175.105 (4/1/91)*

9.0 MONITORING AND ANALYSIS METHODS

Sampling Procedures:

1 NIOSH Method S210. Analyte: Triphenyl phosphate. Matrix: Air. Procedure: Filter collection, extraction with ether. Flow Rate: 1.5 l/min. Sample Size: 100 liters. [Peer Reviewed] *U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual ofAnalytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1*

2 If phosphorus forms are to be differentiated, filter sample immediately after collection. Preserve by freezing at or below -10 deg C. Add 40 mg mercury chloride/l to the samples. ... Do not add either acid or chloroform as a preservative when phosphorus forms are to be determined. /Phosphorus forms/ [Peer Reviewed] *Franson MA (Ed); Standard Methods for the Examination of Water and Wastewater 16th edition p.441 (1985)*

3 NIOSH Method 7905. Analyte: Phosphorus. Matrix: Air. Sampler: Solid sorbent tube (Tenax Gas chromatography, 100 mg/50 mg). Flow Rate: 0.01 to 0.2 l/min: Sample Size: 12 liters. Shipment: Routine. Sample Stability; 94% recovery after 7 days at 25 deg C. /Phosphorus/ [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSHManual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washi*

4 NIOSH Method 7300. Analyte: Phosphorus. Matrix: Air. Sampler: Filter (0.8 um cellulose ester membrane) Flow Rate: 1 to 4 l/min: Sample Size: 500 liters. Shipment: Routine. Sample Stability; Stable. /Phosphorus/ [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSHManual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washi*

Analytical Laboratory Methods:

1 NIOSH Method S210. Analyte: Triphenyl phosphate. Matrix: Air. Procedure: GC/flame ionization detector. Method Evaluation: Method was validated over the range of 1.25 to 6.99 mg/cu m using a 100 liter sample. Method detection limit: not given. Precision (CVT): 0.066 Applicability: Under the conditions of sample size (100 l) the useful range is 0.5 to 9 mg/cu

Applicability: Under the conditions of sample size (100 l) the useful range is 0.5 to 9 mg/cu m. Interferences: No specific interferences. [Peer Reviewed] *U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual ofAnalytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1*

2 A METHOD FOR DETERMINING THE CONTENT IN AIR OF TRIPHENYL PHOSPHATE IS BASED ON ITS HYDROLYSIS IN AN ALKALINE MEDIUM TO FORM PHENOL, WHICH IS THEN COMBINED WITH DIAZOTIZED P-NITROANILINE. THE AZO CMPD FORMED AFTER DIAZOTIZATION, 4-HYDROXY-4-NITROBENZENE, HAS AN INTENSE RED COLOR, WHICH CAN BE DETERMINED VISUALLY OR PHOTOMETRICALLY. [Peer Reviewed] *DRUYAN EA; GIG SANIT (9): 76 (1975)*

3 AN XAD-2 SCREENING METHOD DEVELOPED PREVIOUSLY FOR ORGANOPHOSPHORUS PESTICIDES WAS EXTENDED TO DETERMINE TRIALKYL/ARYL PHOSPHATES IN DRINKING WATER AT THE NG/L LEVEL. RECOVERY STUDIES AT 1, 10, & 100 NG/L LEVELS WERE CARRIED OUT BY FORTIFICATION ONTO XAD-2 RESIN & BY DIRECT ON-STREAM FORTIFICATION OF DRINKING WATER. RECOVERIES WERE GREATER THAN 70% FOR TRIPHENYL PHOSPHATE. [Peer Reviewed] *LEBEL GL ET AL; J ASSOC OFF ANAL CHEM 64 (4): 991 (1981)*

4 SEVERAL TECHNIQUES WERE EVALUATED FOR EXTRACTING TRIPHENYL PHOSPHATE FROM FISH & SEDIMENT SAMPLES. EXTRACTS OF FISH SAMPLES WERE CLEANED UP BY GEL PERMEATION CHROMATOGRAPHY/ALUMINA COLUMN CHROMATOGRAPHY; SEDIMENT EXTRACTS RECEIVED ALUMINA TREATMENT ONLY. CMPD WERE DETERMINED BY GLC WITH NITROGEN-PHOSPHORUS DETECTION. RECOVERIES OF TRIPHENYL PHOSPHATE FROM FORTIFIED RIVER WATER (0.5, 5.0 & 50 UG/L), BY SHAKING WITH DICHLOROMETHANE, RANGED FROM 91-118%. [Peer Reviewed] *MUIR D CG ET AL; J ASSOC OFF ANAL CHEM 64 (1): 79 (1981)*

5 Phosphorus was determined by a continuous flow method using fluorescence quenching of Rhodamine 6G with molybdophosphate. /Phosphorus/ [Peer Reviewed] *Motomizu S et al; Bunseki Kagaku 33 (2): 116-9 (1984)*

6 Phosphorus and organic carbon was simultaneously determined in wastewater using flame ionization and photometric detectors. /Phosphorus/ [Peer Reviewed] *Nakajima K; Water Res 18 (5): 555-9 (1984)*

7 Phosphorus was determined in natural waters by direct current plasma atomic emission spectrometry. /Phosphorus/ [Peer Reviewed] *Urasa IT; Anal Chem 56 (6): 904-8 (1984)*

8 Proton-induced x-ray emission analysis constitutes a method for trace element analysis characterized by multielement capability, detection limits in the low ppm-range, and size resolution approximating a millimicron. /Phosphorus/ [Peer Reviewed] *Malmquist KG et al; Scanning Electron Microsc (4): 1815-26 (1983)*

9 Microamounts of phosphorus in wastewater were determined by high-speed liquid chromatography. /Phosphorus/ [Peer Reviewed] *Sakurai N et al; Fresenius' Z Anal Chem 314 (7): 634-7 (1983)*

10 Fly ash particle sizes are compared using scanning electron microscopy and x-ray elemental analysis. /Phosphorus/ [Peer Reviewed] *Kaufherr N, Lichtman D; Environ Sci Technol 18 (7): 544-7 (1984)*

11 The sample is collected and extracted. A 1 ml aliquot of the decant from the first or second 10 ml portion is placed in a beaker. A 5 ml volume of concentrated nitric acid is added and the mixture evaporated to 1 ml in a fume hood. A 2 ml volume of water is added and the mixture transferred to a test tube. A 1 ml volume of 1M ammonium nitrate and 2 ml of 0.5M ammonium molybdate are added. The solution is heated to boiling and the formation of a yellow precipitate of ammonium phosphomolybdate indicates phosphate. /Phosphate/ [Peer Reviewed] *Welcher FJ, Hahn RB; Semimicro Qual Anal 458 pp (1955) as cited in Environment Canada; Tech Info for Problem Spills: Phosphoric acid (Draft) p.82 (1981)*

12 A range of 1 to 20 mg/l of phosphoric acid as phosphate may be determined colorimetrically. A 35 ml aliquot or less of sample is placed in a 50 ml volumetric flask. A 10 ml volume of vanadate-molybdate reagent is added and the volume taken to mark with distilled water. The

vanadate-molybdate reagent is added and the volume taken to mark with distilled water. The treated sample is left to develop for 10 min, then the absorbance determined at 400 nm using a suitable spectrophotometer with matched 1 cm cells. A blank determination must also be made. The phosphate is determined using a calibration curve. /Phosphate/ [Peer Reviewed] *AWWA; Standard Methods for the Examination of Water and Wastewater, 15 ed (1981) as cited in Environment Canada; Tech Info for Problem Spills: Phosphoric acid (Draft) p.83 (1981)*

13 NIOSH Method 7905. Analyte: Phosphorus. Matrix: Air. Procedure: Gas chromatography, phosphorus flame photometric detector. For phosphorus this method has an estimated detection limit of 0.005 ug/sample. The precision/RSD is 0.024 at 0.6 to 2.4 ug/sample and the recovery is not given. Applicability: The working range is 0.04 to 0.18 mg/cu m (0.008 to 0.16 ppm) for a 12-liter air sample. Interferences: None identified. /Phosphorus/ [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSHManual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washi*

14 NIOSH Method 7300. Analyte: Phosphorus. Matrix: Air. Procedure: Inductively coupled argon plasma. For phosphorus this method has an estimated detection limit of 1 ug/sample. The precision/RSD and the recovery are not given. Applicability: The working range of this method is 0.005 to 2.0 mg/cu m in a 500-liter air sample. Interferences: Spectral interferences. /Phosphorus/ [Peer Reviewed] *U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSHManual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washi*

15 Method 4500-Phosphorus C. Vanadomolybdophosphoric Acid Colorimetric Method. This method determines the total phosphorus in natural waters and wastewaters following a digestion procedure to release phosphorus as orthophosphate. The method is based on the formation of yellow vanadomolybdo- phosphoric acid upon the addition of ammonium molybdate and vanadium to ortho- phosphate solution. This method is most useful for routine analysis in the range of 1 to 20 mg phosphorus/l. Minimum detectable concentration is 200 ug phosphorus/l in 1-cm spectrometer cells. Positive interference is caused by silica and arsenate. Negative interference is caused by arsenate, fluoride, thorium, bismuth, sulfide, thiosulfate, thiocyanate or excess molybdate. /Phosphorus/ [Peer Reviewed] *Franson MA, ed; Standard Methods for the Examination of Water and Wastewater, 17th ed p.4-173 (1989)*

16 Method 4500-Phosphorus D. Stannous Chloride Method. This method determines total phosphorus in natural waters and wastewaters following a digestion procedure to release phosphorus as orthophosphate. The method is based upon the reduction of molybdophosphoric acid by stannous chloride to intensely colored molybdenum blue. This method is most suited for the concentration range of 0.01-6 mg phosphorus/l. The minimum detectable concentration is 3 ug phosphorus/l. The sensitivity at 0.3010 absorbance is 10 ug phosphorus/l for an absorbance change of 0.009. Positive interference is caused by silica and arsenate. Negative interference is caused by arsenate, fluoride, thorium, bismuth, sulfide, thiosulfate, thiocyanate, or excess molybdate. /Phosphorus/ [Peer Reviewed] *Franson MA, ed; Standard Methods for the Examination of Water and Wastewater, 17th ed p. 4-175 (1989)*

10.0 ADDITIONAL REFERENCES

Special Reports:

WHO; Diseases Caused by Phosphorus and Its Toxic Compounds; Early Detection of Occupational Diseases pg 53-62 (1986). Review ofdiseases and health related effects resulting from exposure to phosphorus or phosphorus cmpd. /Phosphorus or phosphorus cmpd/

Triphenyl phosphate

NIOSH Pocket Guide

1.0 CHEMICAL NAME, STRUCTURE/FORMULA, CAS AND RTECS NOS., AND DOT ID AND GUIDE NOS.

Chemical Name: Triphenyl phosphate
Formula: $(C_6H_5O)_3PO$
Structure/Formula: $(C_6H_5O)_3PO$
CAS Number: 115-86-6
RTECS Number: TC8400000

2.0 SYNONYMS, TRADENAMES AND CONVERSION FACTORS

Synonyms/Tradenames: Phenyl phosphate; TPP; Triphenyl ester of phosphoric acid

3.0 EXPOSURE LIMITS (TWA UNLESS NOTED OTHERWISE)

NIOSH Recommended Exposure Limits (RELs): Time-weighted average (TWA) 3 mg/m^3
OSHA Permissible Exposure Limits (PELs): Time-weighted average (TWA) 3 mg/m^3

4.0 IDLH

1000 mg/m^3

5.0 PHYSICAL DESCRIPTION

Colorless, crystalline powder with a phenol-like odor.

6.0 CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 326.3
Boiling Point: 776°F
Solubility in Water: (129°F) 0.002%
Flash Point: 428°F
Ionization Potential: Unknown
Specific Gravity: 1.29
Flammability Class: Combustible Solid
Vapor Pressure: (380°F) 1 mmHg
Freezing Point: 120°F
Upper Explosive Limit in air (% by volume): Unknown
Lower Explosive Limit in air (% by volume): Unknown

7.0 INCOMPATIBILITIES AND REACTIVITIES

None reported

8.0 MEASUREMENT METHOD (SEE TABLE 1)

Particulate filter; Diethyl ether; Gas Chromatography with flame photometric detection for sulfur, nitrogen,

or phosphorus; IV [#5038]

9.0 PERSONAL PROTECTION AND SANITATION (SEE TABLE 3)

Skin: No recommendation is made specifying the need for personal protective equipment for the body.
Eyes: No recommendation is made specifying the need for eye protection.
Wash Skin: No recommendation is made specifying the need for washing the substance from the skin (either immediately or at the end of the work shift).
Remove: No recommendation is made specifying the need for removing clothing that becomes wet or contaminated.
Change: No recommendation is made specifying the need for the worker to change clothing after the workshift.

10.0 RECOMMENDATIONS FOR RESPIRATOR SELECTION (SEE TABLE 4)

NIOSH/OSHA

15 mg/m^3: D

30 mg/m^3: Any air-purifying respirator with a high-efficiency particulate filter.(Assigned Protection Factor = 10)/Any dust respirator except single-use and quarter-mask respirators.(Assigned Protection Factor = 10)/Any supplied-air respirator.(Assigned Protection Factor = 10)

75 mg/m^3: Any supplied-air respirator operated in a continuous-flow mode.(Assigned Protection Factor = 25)/Any powered, air-purifying respirator with a dust and mist filter.(Assigned Protection Factor = 25)

150 mg/m^3: Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter.(Assigned Protection Factor = 50)/Any supplied-air respirator that has a tight-fitting facepiece and is operated in a continuous-flow mode.(Assigned Protection Factor = 50)/Any powered, air-purifying respirator with a tight-fitting facepiece and a high-efficiency particulate filter.(Assigned Protection Factor = 50)/Any self-contained breathing apparatus with a full facepiece.(Assigned Protection Factor = 50)/Any supplied-air respirator with a full facepiece.(Assigned Protection Factor = 50)

1000 mg/m^3: Any supplied-air respirator operated in a pressure-demand or other positive-pressure mode.(Assigned Protection Factor = 2000)

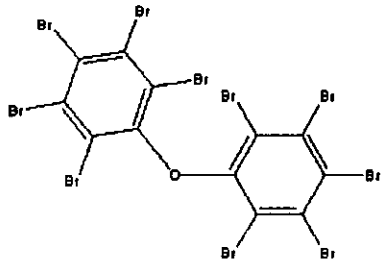
Emergency or planned entry into unknown concentrations or IDLH conditions: Any self-contained breathing apparatus that has a full facepiece and is operated in a pressure-demand or other positive-pressure mode(Assigned Protection Factor = 10000)/Any supplied-air respirator that has a full facepiece and is operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive-pressure mode.(Assigned Protection Factor = 10000)Escape: Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter.(Assigned Protection Factor = 50)/Any appropriate escape-type, self-contained breathing apparatus.

11.0 HEALTH HAZARDS

Exposure Routes: Inhalation, Ingestion
Exposure Symptoms (See Table 5): Minor changes in blood enzymes; in animals: Muscle Weakness, Paralysis
Breath (See Table 6): If a person breathes large amounts of this chemical, move the exposed person to fresh air at once. If breathing has stopped, perform mouth-to-mouth resuscitation. Keep the affected person warm and at rest. Get medical attention as soon as possible.
Swallow (See Table 6): If this chemical has been swallowed, get medical attention immediately.

Swallow (See Table 6): If this chemical has been swallowed, get medical attention immediately.
Target Organs (See Table 5): Blood, Peripheral nervous system

TR-309
Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) In F344/N Rats and B6C3F$_1$ Mice (Feed Studies)



Chemical Formula: $C_{12}Br_{10}O$ - 3D Structure

Toxicology and carcinogenesis studies of decabromodiphenyl oxide, a flame retardant for plastics and other materials, were conducted by exposing groups of 50 male and 50 female F344/N rats and B6C3F$_1$ mice at 0, 25,000, and 50,000 ppm in the diet for 103 weeks. These concentrations were selected because no toxicity was observed at any dose in the 14-day or 13-week studies and 50,000 ppm chemical in the diet is considered to be the highest dose to which rats and mice can be exposed for extended periods of time without reducing the nutritional value of the diet. No compound-related gross or microscopic pathologic effects were observed in the 14-day or 13-week studies.

Body weights of dosed male and female rats and mice in the 2-year studies were comparable to those of the controls. Decreased survival of low dose male rats was not believed to be compound related. No other effects on survival were observed in the 2-year studies. Loss of control male mice (presumably due to fighting) was significant during the first part of the study.

In the 2-year studies, nonneoplastic lesions were observed at increased incidences in rats and mice of each sex. Thrombosis and degeneration of the liver, fibrosis of the spleen, and lymphoid hyperplasia were observed in high dose male rats. Degeneration of the eye was observed in low dose female rats. Nonneoplastic lesions observed in dosed mice were granulomas in the liver of low dose males and hypertrophy in the liver of low dose and high dose males. Follicular cell hyperplasia was observed in thyroid glands of dosed male mice (control, 2/50; low dose, 10/50; high dose, 19/50).

The incidences of neoplastic nodules in the liver of low and high dose male rats (1/50; 7/50; 15/49) and high dose female rats (1/50; 3/49; 9/50) were significantly greater than those in the controls. Mononuclear cell leukemia occurred in dosed male rats with a positive trend (30/50; 33/50; 35/50); this marginal increase was not considered biologically significant. Acinar cell adenomas were observed in the pancreas of four high dose male rats, and a sarcoma was observed in the spleen of one low dose and one high dose male rat. Hepatocellular adenomas or carcinomas (combined) occurred at marginally increased incidences in dosed male mice (8/50; 22/50; 18/50). The incidences of thyroid gland follicular cell adenomas or carcinomas (combined) were increased in dose male mice (0/50; 4/50; 3/50).

A study of decabromodiphenyl oxide absorption from the gastrointestinal tract indicated that absorption was minimal, possibly less than 1%, at the doses administered in the 2-year studies. Additional chemical analysis indicated than the decabromodiphenyl oxide used in these studies contained several less brominated diphenyl oxides. Therefore, since absorption and toxicity of minor impurities are unknown, effects observed in these studies must be attributed to the approximately 95% pure preparation used rather than to pure decabromodiphenyl oxide.

Decabromodiphenyl oxide was not mutagenic in strains TA1535, TA1537, TA98, or TA100 of *Salmonella typhimurium* in the presence or absence of Aroclor 1254-induced Sprague-Dawley male rat or Syrian hamster liver S9 when tested according to the preincubational protocol. Decabromodiphenyl

oxide was not mutagenic in the mouse lymphoma L5178Y/TK$^{+/-}$ assay in the presence or absence of Aroclor 1254-induced F344/N male rat liver S9. Decabromodiphenyl oxide did not induce sister-chromatid exchanges or chromosomal aberrations in Chinese hamster ovary cells in vitro in the presence or absence of S9 prepared from livers of Aroclor 1254-induced male Sprague-Dawley rats.

An audit of experimental data was conducted for these 2-year studies on decabromodiphenyl oxide. No data discrepancies were found that influenced the final interpretations.

Under the conditions of these 2-year feed studies of decabromodiphenyl oxide, there was *some evidence of carcinogenicity* for male and female F344/N rats as shown by increased incidences of neoplastic nodules of the liver in low dose (25,000 ppm) males and high dose (50,000 ppm) groups of each sex. There was *equivocal evidence of carcinogenicity* for male B6C3F1 mice as shown by increased incidences of hepatocellular adenomas or carcinomas (combined) in the low dose group and of thyroid gland follicular cell adenomas or carcinomas (combined) in both dosed groups. There was *no evidence of carcinogenicity* for female B6C3F1 mice receiving 25,000 or 50,000 ppm in the diet. Several nonneoplastic lesions were observed at increased incidences, the most notable being thyroid gland follicular cell hyperplasia in male mice.

Synonyms: decabromodiphenyl ether; bis(pentabromophenyl)ether; DBDPO

Target Organs & Incidences from 2-year Studies

Report Date: May 1986

NTIS# PB86-247780/AS

Target Organs and Levels of Evidence for NTP Technical Report Number 309
Produced from Chemtrack Database 07/23/96

CHEMICAL/ CAS NUMBER	PEER REVIEW DATE	PRIMARY USES	ROUTE/EXPOSURE LEVELS	STUDY LABORATORY
DECABROMODIPHENYL OXIDE 1163-19-5	08/14/85	FLAME RETARDANT. FOR NITRILE RESINS, POLYESTER AND TEXTILE COATINGS. (TDB)	Dosed-Feed 0,25000,50000 PPM/50 PER GROUP	Hazleton (Vienna)

LEVELS OF EVIDENCE OF CARCINOGENICITY--ORGAN/TISSUE (NEOPLASM):

MR: SOME EVIDENCE	LIVER: NEOPLASTIC NODULE 1/50 7/50 15/49
FR: SOME EVIDENCE	LIVER: NEOPLASTIC NODULE 1/50 3/49 9/50
MM: EQUIVOCAL EVIDENCE	LIVER: ADENOMA 4/50 12/50 12/50 OR CARCINOMA 5/50 14/50 8/50 COMBINED 8/50 22/50 18/50
	THYROID GLAND: FOLLICULAR CELL ADENOMA 0/50 3/50 3/50 OR FOLLICULAR CELL CARCINOMA 0/50 1/50 0/50 COMBINED 0/50 4/50 3/50
NON-NEOPLASTIC LESIONS:	THYROID GLAND: HYPERPLASIA, FOLLICULAR CELL
FM: NO EVIDENCE	

Ether, bis(pentabromophenyl)

RTECS - Registry of Toxic Effects of Chemical Substances

Document Outline

1.0 SUBSTANCE IDENTIFICATION

RTECS Number: KN3525000
Chemical Name: Ether, bis(pentabromophenyl)
CAS Number: 1163-19-5
Molecular Formula: C12-Br10-O
Molecular Weight: 959.22
Substance Investigated as: Tumorigen, Reproductive Effector
Last Revision Date: 1997

2.0 SYNONYM(S)/TRADENAME(S)

1 1,1'-Oxybis(2,3,4,5,6-pentabromobenzene)
2 Benzene, 1,1'-oxybis(2,3,4,5,6-pentabromo- (9CI)
3 Berkflam B 10E
4 Bis(pentabromophenyl)ether
5 BR 55N
6 Bromkal 82-ode
7 Bromkal 83-10DE
8 Dbdpo
9 De 83R
10 Decabrom
11 Decabromobiphenyl ether
12 Decabromobiphenyl oxide
13 Decabromodiphenyl oxide
14 Decabromophenyl ether
15 Ether, decabromodiphenyl
16 FR 300
17 FR 300BA
18 FRP 53
19 NCI-C55287
20 Pentabromophenyl ether
21 Saytex 102
22 Saytex 102E
23 Tardex 100

3.0 HEALTH HAZARD DATA

ACUTE TOXICITY

OTHER LD/LC - OTHER LETHAL DOSE/CONC

Rat

LD - ROUTE: Oral; **DOSE:** >2 gm/kg
LD - ROUTE: Skin; **DOSE:** >3 gm/kg

REPRODUCTIVE EFFECTS

Rat

ROUTE: Oral; **DOSE:** 100 mg/kg; **DURATION:** female 6-15D of pregnancy
TOXIC EFFECTS:
Effects on Fertility - Post-implantation mortality (e.g., dead and or resorbed implants per total number of implants)

TUMORIGENIC EFFECTS

Rat

ROUTE: Oral; **DOSE:** 1092 gm/kg/2Y continuous
TOXIC EFFECTS:
Tumorigenic - Neoplastic by RTECS criteria
Liver - Tumors

OTHER MULTIPLE DOSE TOXICITY DATA

Rat

ROUTE: Oral; **DOSE:** 24 gm/kg/30D continuous
TOXIC EFFECTS:
Liver - Other changes
Endocrine - Thyroid weight (goiter)

4.0 STANDARDS AND REGULATIONS

OEL-RUSSIA:STEL 3 mg/m3 JAN 1993

5.0 NIOSH DOCUMENTS

National Occupational Exposure Survey 1983: Hazard Code X4046; Number of Industries 12; Total Number of Facilities 981; Number of Occupations 30; Total Number of Employees 32661; Total Number of Female Employees 8875

6.0 REVIEWS

1 IARC Cancer Review:Animal Limited Evidence
2 IARC Cancer Review:Group 3

7.0 STATUS IN U.S.

1 EPA TSCA Section 8(b) CHEMICAL INVENTORY
2 EPA TSCA Section 8(d) unpublished health/safety studies

2 EPA TSCA Section 8(d) unpublished health/safety studies
3 EPA TSCA TEST SUBMISSION (TSCATS) DATA BASE, SEPTEMBER 1997
4 NTP Carcinogenesis Studies (feed);equivocal evidence:mouse NTPTR* NTP-TR-309,86
5 NTP Carcinogenesis Studies (feed);some evidence:rat NTPTR* NTP-TR-309,86
6 On EPA IRIS database

DECABROMOBIPHENYL ETHER

HSDB - Hazardous Substances Data Bank

0.0 ADMINISTRATIVE INFORMATION

Hazardous Substances Data Bank Number: 2911
Last Revision Date: 970423
Review Date: Reviewed by SRP on 2/28/92
Update History:

1 Complete Update on 04/23/97, 1 field added/edited/deleted.
2 Complete Update on 01/27/97, 1 field added/edited/deleted.
3 Complete Update on 05/11/96, 1 field added/edited/deleted.
4 Complete Update on 01/26/96, 1 field added/edited/deleted.
5 Complete Update on 12/30/94, 1 field added/edited/deleted.
6 Complete Update on 11/15/94, 1 field added/edited/deleted.
7 Complete Update on 09/13/94, 2 fields added/edited/deleted.
8 Complete Update on 06/14/94, 1 field added/edited/deleted.
9 Complete Update on 05/05/94, 1 field added/edited/deleted.
10 Complete Update on 03/25/94, 1 field added/edited/deleted.
11 Complete Update on 09/02/93, 1 field added/edited/deleted.
12 Complete Update on 06/04/93, 43 fields added/edited/deleted.
13 Field update on 12/27/92, 1 field added/edited/deleted.
14 Field Update on 09/03/92, 1 field added/edited/deleted.
15 Complete Update on 09/26/91, 1 field added/edited/deleted.
16 Complete Update on 10/22/90, 2 fields added/edited/deleted.
17 Complete Update on 09/23/88, 1 field added/edited/deleted.

1.0 SUBSTANCE IDENTIFICATION

Name of Substance: DECABROMOBIPHENYL ETHER
CAS Registry Number: 1163-19-5
Synonyms:

1 BENZENE, 1,1'-OXYBIS(2,3,4,5,6-PENTABROMO- [Peer Reviewed]
2 BERKFLAM B 10E [Peer Reviewed]
3 BIS(PENTABROMOPHENYL) ETHER [Peer Reviewed]
4 BR 55N [Peer Reviewed]
5 BROMKAL 82-0DE [Peer Reviewed]
6 BROMKAL 83-10DE [Peer Reviewed]
7 DECABROMOBIPHENYL OXIDE [Peer Reviewed]
8 DECABROMODIPHENYL ETHER [Peer Reviewed]
9 DECABROMODIPHENYL OXIDE [Peer Reviewed]
10 DECABROMOPHENYL ETHER [Peer Reviewed]
11 ETHER, BIS(PENTABROMOPHENYL) [Peer Reviewed]
12 FR 300BA [Peer Reviewed]
13 FRP 53 [Peer Reviewed]
14 PENTABROMOPHENYL ETHER [Peer Reviewed]

Molecular Formula: C12-BR10-O [Peer Reviewed]
RTECS Number: NIOSH/KN3525000

2.0 MANUFACTURING/USE INFORMATION

Methods of Manufacturing:

BROMINATION OF DIPHENYL OXIDE IN THE PRESENCE OF A FRIEDEL-CRAFTS CATALYST. [Peer Reviewed] *Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V10 385 (1980)*

Formulations/Preparations:

1 Technical product composition: decabromodiphenyl oxide, 77.4%, Nonabromodiphenyl oxide, 21.8%, Octabromodiphenyl oxide, 0.8%. [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.19 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*

2 Usually contains more than 85 wt% decabromodiphenyl oxide. [Peer Reviewed] *Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V10 385 (1980)*

Manufacturers:

1 Ethyl Corp, Hq, 330 S Fourth St, PO Box 2189, Richmond, VA 23217, (804) 788-5000; Chemicals Group, Ethyl Tower, 451 Florida Blvd, Baton Rouge, LA 70801; Production site: Magnolia, AR 71753 [Peer Reviewed] *SRI. 1992 Directory of Chemical Producers-United States of America. Menlo Park, CA: SRI International, 1992. 552*

2 Great Lakes Chemical Corporation, Hq, Highway 52, North West, PO Box 2200, West Lafayette, IN 47906, (317) 497-6100; Production site: El Dorado, AR 71730 [Peer Reviewed] *SRI. 1992 Directory of Chemical Producers-United States of America. Menlo Park, CA: SRI International, 1992. 552*

Other Manufacturing Information:

Often plastic additives may be released by leaching. However experiments performed with acrylonitrile butadiene styrene and acrylonitrile butadiene styrene polystyrene polymers containing decabromodiphenyl ether into water at room and elevated temperatures and into 3% acetic acid at elevated temperatures resulted in no leaching at a detection limit of 0.5 ppm of bromine. [Peer Reviewed] *Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973)*

Major Uses:

Decabromodiphenyl oxide is an unreactive, additive flame retardant widely used for its thermal stability and its low cost in thermoplastic resins, thermoset resins, textiles and adhesives. The major applications are in high impact polystyrene, glass reinforced thermoplastic polyester and moulding resins, low density polyethylene extrusion coatings, polypropylene (homo- and copolymers), acrylonitrile butadiene styrene rubber, nylon and polyvinyl chloride. [Peer Reviewed] *IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V48 75 (1990)*

Consumption Patterns:

ESSENTIALLY 100% AS FLAME RETARDANT [Peer Reviewed] *SRI*

U.S. Production:

1 (1973) MORE THAN 5X10+5 G [Peer Reviewed] *SRI*

2 (1975) MORE THAN 5X10+5 G [Peer Reviewed] *SRI*

3.0 CHEMICAL AND PHYSICAL PROPERTIES

Color/Form: White to off-white powder [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.18 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*
Melting Point: 295-305 deg C [Peer Reviewed] *Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V10 385 (1980)*
Molecular Weight: 959.12 [Peer Reviewed]
Density/Specific Gravity: 3.0 [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.19 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*
Octanol/Water Partition Coefficient: log Kow= 5.24 [Peer Reviewed] *Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973)*
Solubilities: Water: 20-30 ppb; Cottonseed oil: 600 ppm, acetone: 500 ppm, chlorobenzene: 6,000 ppm, o-xylene: 8,700 ppm. [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.19 (1986)Technical Rpt Series No. 309 NIH Pub No. 86-2565*
Vapor Pressure: 5 mm Hg @ 306 deg C [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.18 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*
Other Chemical/Physical Properties:

1 Decomposition point: Ca 425 deg C [Peer Reviewed] *Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V10 385 (1980)*
2 Henry's Law constant: 4.45X10-8 atm-cu m/mol (est). [Peer Reviewed] *Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991)*

5.0 TOXICITY/BIOMEDICAL EFFECTS

SUMMARY

Evidence for Carcinogenicity:

1 Evaluation: There is limited evidence for the carcinogenicity of decabromodiphenyl oxide in experimental animals. No data were available from studies in humans on the carcinogenicity of decabromodiphenyl oxide. Overall evaluation: Decabromodiphenyl oxide is not classifiable as to its carcinogenicity to humans (Group 3). [QC Reviewed] *IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. 48 82 (1990)*
2 CLASSIFICATION: C; possible human carcinogen. BASIS FOR CLASSIFICATION: Based on no human data and limited evidence of carcinogenicity in animals; namely, significantly increased incidences of neoplastic liver nodules in male and female rats and increased incidences of hepatocellular adenomas or carcinomas (combined) in male mice. HUMAN CARCINOGENICITY DATA: None. ANIMAL CARCINOGENICITY DATA: Limited. [QC Reviewed] *U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) on Decabromodiphenyl ether (DBOPE) (1163-19-5) from the National Library of Medicine's TOXNET System, November 1, 1994*

TOXICITY EXCERPTS

Human Toxicity Excerpts:

Thyroid and reproductive dysfunction was investigated in workers exposed for at least 240 hr to decabromobiphenyl and decabromobiphenyloxide over a 4 yr period. The average period of

decabromobiphenyl and decabromobiphenyloxide over a 4 yr period. The average period of employment was 3.9 yr. Air samples of decabromobiphenyl in the manufacturing area showed 0.18 to 0.23 mg/cu m for an 8 hr time weighted average. Of 18 workers exposed for 3 yr or longer, thyroid nodules were seen in 3; another nodule was seen in a worker exposed to high chlorine concentrations. Significant correlation was seen between length of employment and concentrations of follicle stimulating hormone in workers exposed to decabromobiphenyl. An abnormal follicle stimulating hormone value was found in only one worker. A testicular cyst was found in one exposed worker, and epididymal nodules in two others. No testicular or epididymal nodules were seen among comparisons. No detectable decarbromobiphenyl was found in serum. It was concluded that thyroid hyperplasia may result from exposure to decarbromobiphenyl, but no definite statement can be made concerning adverse effects on the prevalence of testicular and epididymal nodules because of their prevalence in the general population. [Peer Reviewed] *Bialik O; NIOSH; Endocrine Function Of Workers Exposed To PBB And PBBO. Terminal Progress Report. Grant No. I-RO1-OH-01034-01 63 (1982)*

Non-Human Toxicity Excerpts:

1 30-DAY DIETARY FEEDING STUDY IN RATS ESTABLISHED 8 MG DECARBROMOBIPHENYL OXIDE/KG/DAY AS UNEQUIVOCAL NO EFFECT LEVEL. REPRODUCTIVE CAPACITY OF RATS WAS NOT AFFECTED AT 3, 30 OR 100 MG DECARBROMOBIPHENYL OXIDE/KG/DAY. NO EFFECTS OBSERVED ON CYTOGENETIC EXAM OF BONE MARROW CELLS FROM REPRODUCTIVE STUDY. [Peer Reviewed] *NORRIS JM ET AL; ENVIRON HEALTH PERSPECT (11): 153-61 (1975)*

2 DECABROMODIPHENYL OXIDE HAS LOW ACUTE ORAL TOXICITY & LOW SKIN ABSORPTION TOXICITY. 30-DAY DIETARY FEEDING STUDY IN RATS ESTABLISHED 80 MG/KG/DAY AS MARGINAL EFFECT LEVEL. [Peer Reviewed] *NORRIS JM ET AL; ENVIRON HEALTH PERSPECT (11): 153-61 (1975)*

3 Daily intubation of pregnant female rats on gestations days 6-15 with 0, 10, 100, or 1,000 mg decabromodiphenyl oxide/kg, suspended in corn oil, caused no teratogenic response. Some fetal toxicity was observed in these studies in the form of subcutaneous edema and delayed ossification of normally developed bones of the fetal skull. These effects were observed at the high dose only. [Peer Reviewed] *DHHS/NTP; Toxicology & Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.19 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*

4 Decabromodiphenyl oxide has low acute toxicity. Oral administration of doses up to 2,000 mg/kg as a 10% suspension in corn oil failed to produce any signs of toxicity in rats either directly after dosing or during a 14 day observation period. This chemical is not a dermal irritant to rats or rabbits and is only mildly irritating when placed in the eyes of rabbits. Repeated oral doses of up to 800 mg/kg per day produced no overt indication of toxicity during a 30 day study. [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.19 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*

5 NTP studies of decabromodiphenyl oxide mutagenicity indicate that it was not mutagenic in Salmonella typhimurium strains TA1535, TA1537, TA98, or TA100 in the presence or absence of Aroclor 1254 induced male Sprague Dawley rat or male Syrian hamster liver S9 when tested according to the preincubation protocol. It was also not mutagenic in the mouse lymphoma L5178y/TK + or - assay in the presence or absence of Aroclor 1254 induced male F344 liver S9. Tests for cytogenetic effects in Chinese hamster ovary cells indicated that this chemical does not cause chromosomal aberrations or sister chromatid exchanges either in the presence or absence of S9 prepared from livers of Aroclor 1254 induced male Sprague Dawley rats. [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.20 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*

TOXICITY VALUES

National Toxicology Program Reports:

... Carcinogenesis studies of decabromodiphenyl oxide were conducted by exposing groups of 50 male and 50 female F344/N rats and B6C3F1 mice at 0, 25,000 and 50,000 ppm in the diet for 103 weeks. ... The incidences of neoplastic nodules in the liver of low and high dose male rats (1/50; 7/50; 15/49) and high dose female rats (1/50; 3/49; 9/50) were significantly greater than those in the controls. Mononuclear cell leukemia occurred in dosed male rats with a positive trend (30/50; 33/50; 35/50); this marginal increase was not considered biologically significant. Acinar cell adenomas were observed in the pancreas of four high dose male rats, and a sarcoma was observed in the spleen of one low dose and one high dose male rat. Hepatocellular adenomas or carcinomas (combined) occurred at marginally increased incidences in dosed male mice (8/50; 22/50; 18/50). The incidences of thyroid gland follicular cell adenomas or carcinomas (combined) were increased in dosed male mice (0/50; 4/50; 3/50). Under the conditions of these 2 yr studies ... there was some evidence of carcinogenicity for male and female F344/N rats as shown by increased incidences of neoplastic nodules of the liver in low dose (25,000 ppm) males and high dose (50,000 ppm) groups of each sex. There was equivocal evidence of carcinogenicity for male B6C3F1 mice as shown by increased incidences of hepatocellular adenomas or carcinomas (combined) in the low dose group and of thyroid gland follicular cell adenomas or carcinomas (combined) in both dosed groups. There was no evidence of carcinogenicity for female B6C3F1 mice receiving 25,000 or 50,000 ppm in the diet. ... [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.11 (1986) Technical Rpt Series No. 309 NIH Pub No. 86-2565*

IARC Summary and Evaluation:

Evaluation: There is limited evidence for the carcinogenicity of decabromodiphenyl oxide in experimental animals. No data were available from studies in humans on the carcinogenicity of decabromodiphenyl oxide. Overall evaluation: Decabromodiphenyl oxide is not classifiable as to its carcinogenicity to humans (Group 3). [QC Reviewed] *IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. 48 82 (1990)*

PHARMACOKINETICS

Absorption, Distribution and Excretion:

1 AFTER ADMIN OF (14)C DECABROMODIPHENYL OXIDE BY INTRAGASTRIC INTUBATION, ALL (14)C ACTIVITY WAS ELIMINATED BY WAY OF FECES WITHIN 2 DAYS. [Peer Reviewed] *NORRIS JM ET AL; ENVIRON HEALTH PERSPECT (11): 153-61 (1975)*

2 Studies with (14)C labeled decabromodiphenyl oxide administered orally to Sprague Dawley rats indicate that more than 99% of the administered label was excreted in feces within 2 days following administration. An analysis of bromine in tissues following long term exposure in diets that provided 0.1 mg/kg per day to rats indicated a slight increase in bromine content in liver and adipose tissue at 90 days but no significant increase following 12 months of exposure. A significant increase in the bromine content of adipose, but no other tissues, was observed following a similiar dose of decabromodiphenyl oxide for 2 years but not at lower doses. There was no indication as to whether the failure of decabromodiphenyl oxide to accumulate in tissues was due to lack of absorption from the gastrointestinal tract or rapid metabolism and clearance. [Peer Reviewed] *DHHS/NTP; Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) p.18 (1986) Technical Rpt Series No. 309 Pub No. 86-2565*

3 The disposition of 14(C)-labeled decabromobiphenyl ether in male Fischer rats dosed by feeding (0.025-5.0% of the diet) or intravenously (1 mg/kg) was determined. For rats dosed

feeding (0.025-5.0% of the diet) or intravenously (1 mg/kg) was determined. For rats dosed by feeding, intestinal absorption of decarbromobiphenyl ether was evident in that the intact compound was present in extracts of liver. For these rats, the size of the liver increased with increasing concentration of decarbromobiphenyl ether in the diet. Liver contained a maximum of 0.449% of the administered radioactivity at 24 hr after feeding rats a diet containing 0.0277% 14(C) decarbromobiphenyl ether; no other organ or tissue contained more than 0.26%. The total amount of radioactivity found in tissues was less than 1% of the dose. Of the radioactivity recovered in the feeding experiments, more than 99% was in the feces and gut contents at 72 hr; a maximum of 0.012% of the dose was in the urine. In the feces of rats fed 14(C) decabromobiphenyl ether, there were three metabolites, which together comprised 1.5-27.95% of the radioactivity. Since absorption was minimal, most of the metabolism of 14(C) decarbromobiphenyl ether apparently took place in the gastrointestinal tract. The metabolites increased in percent of total radioactivity with the content of decarbromobiphenyl ether in the diet, an indication that enzyme induction in intestinal bacteria may have occurred at the higher doses. More extensive metabolism of 14(C) decarbromobiphenyl ether occurred after intravenous administration; only 37% of the radioactivity in the feces was unchanged decarbromobiphenyl ether. At 72 hr after dosing, fecal excretion accounted for 70% of the dose; only 0.129% appeared in the urine. Muscle retained 12.9% and skin 7.25% of the radioactivity administered. In 4 hr, rats with biliary cannulas excreted 7.17% of the intravenously administered radioactivity in the bile; less than 1% was excreted as intact decarbromçbiphenyl ether. Biliary excretion was apparently the major route for elimination of the intravenously administered compound. The rapid excretion and extensive metabolism of decabromobiphenyl ether, relative to other polyhalogenated compounds, are advantagenous properties that may allow it to be used in place of structurally similar compounds presently employed in industrial applications. [Peer Reviewed] *El Dareer SM et al; J Toxicol Environ Health 22 (4): 405-16 (1987)*

7.0 ENVIRONMENTAL FATE/EXPOSURE POTENTIAL

SUMMARY

Environmental Fate/Exposure Summary:

Decabromodiphenyl ether is used as a flame retardant for plastics, especially high-impact polystyrene, and to treat fabrics forautomobiles and tents. It may be released to the environment inwastewater or dust during its production and use. If released to soil, decabromodiphenyl ether would be expected to adsorb strongly. If released to water, decabromodiphenyl ether would accumulate in the sediment. Photodegradation on the soil surface and surface layers of water may be fairly rapid. Limited data suggests some biodegradation in water. If released to the atmosphere, decabromodiphenyl ether containing dust will be removed by gravitational settling. No bioconcentration in fish has been observed.Exposure will be primarily occupational by inhalation of dust and by dermal contact. (SRC) [Peer Reviewed]

POLLUTION SOURCES

Natural Occurring Sources:

Decabromodiphenyl ether does not occur naturally(1). [Peer Reviewed] *(1) IARC; IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans 48: 73-84 (1989)*

Artificial Sources:

Decabromodiphenyl ether is a fire retardant additive for thermoplastic resins, polyethylene,

polystyrene, adhesives, polyester fibers and coatings for textiles, and paints(1-3,5). It is especially used for high-impact polystyrene such as for television and radio sets and to treat textiles for automobiles and tents(5). Decabromodiphenyl ether may be released in waste water and in dust in connection with its manufacture and use. It is released during the pyrolysis of material containing the flame retardant(4). Plastic additives may be released by leaching. However experiments perform with ABS and ABS-polystyrene polymers containing decabromodiphenyl ether into water at room and elevated temperatures and into 3% acetic acid at elevated temperatures resulted in no leaching at a detection limit of 0.5 ppm of bromine(1). A small amount of leaching was obtained when the polymer was immersed in cottonseed oil at elevated temperatures. [Peer Reviewed] *(1) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (2) Watanabe I et al; Bull Environ Contam Toxicol 36: 839-42 (1986) (3) Thoma H et al; Chemosphere 16: 277-85 (1987) (4) Thoma H, Hutzinger O; Chemosphere 16: 1353-60 (1987) (5) IARC; IARC Monog*

ENVIRONMENTAL FATE

1 TERRESTRIAL FATE: If released to soil, decabromodiphenyl ether would be expected to adsorb strongly. Photodegradation on the soil surface may be fairly rapid. Biodegradability in soil is unknown. (SRC) [Peer Reviewed]

2 AQUATIC FATE: If released in water, decabromodiphenyl ether would adsorb strongly to sediment and particulate matter in the water column. Photodegradation should occur in the surface layer(1-2). Limited data suggests that biodegradation may occur in river water (27% biodegradation in 3 days)(1). No appreciable bioconcentration may occur in fish(1). [Peer Reviewed] *(1) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (2) Watanabe I et al; Bull Environ Contam Toxicol 36: 839-42 (1986) (3) Kondo M et al; Eisei Kagaku 34: 115-22 (1988)*

3 ATMOSPHERIC FATE: If released to the atmosphere, decabromodiphenyl ether would occur as a dust and be removed by gravitational settling. Direct photodegradation may be fairly rapid based upon studies with sunlight irradation(1-2,SRC). [Peer Reviewed] *(1) Norris JM et al; Apply Polymer Sympos 22: 195-219 (1979) (2) Watanabe I et al; Bull Environ Contam Toxicol 36: 839-42 (1986)*

ENVIRONMENTAL TRANSFORMATIONS

Biodegradation:

Judged to be moderate to hard to degrade according to results obtained by the "cultivation method" in which 27% and 4% degradation occurred in 3 days in river water and coastal sea water in Japan(1). [Peer Reviewed] *(1) Kondo M et al; Eisei Kagaku 34: 115-22 (1988)*

Abiotic Degradation:

1 /Decabromobiphenyl oxide/ is degraded by ultraviolet light in the wavelength range and intensity of light. [Peer Reviewed] *DHHS/NTP; Toxicol and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) Report #309 NIH Pub #86-2565 p.19 (1986)*

2 Bromine is released when decabromodiphenyl ether is exposed to sunlight. After 98 days of exposure, the amount of bromine produced is that which corresponds to the breakdown of about 300 times the initial amount of decabromodiphenyl ether soluble in water(1). GC/MS analysis performed at the conclusion of the experiment did not indicate the presence of aromatic bromine compounds. When decabromodiphenyl ether dissolved in octanol was exposed to radiation from a sunlamp, 50% degradation occurred in 4 hr(1). Reductive dehalogenation was the degradative process(1). The products resulting from exposure of a hexane solution of decabromodiphenyl ether to sunlight was the nona-, octa-, hepta-, and hexabrominated compounds(2). However other studies indicate that photohydroxylation is the

hexabrominated compounds(2). However other studies indicate that photohydroxylation is the favored degradative route in aqueous solution and that the hydroxylated degradation products would rapidly decompose. Ethers are generally resistant to hydrolysis(3). [Peer Reviewed] *(1) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (2) Watanabe I et al; Bull Environ Contam Toxicol 36: 839-42 (1986) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods NY: McGraw-Hill Chapt 7 (1982)*

ENVIRONMENTAL TRANSPORT

Bioconcentration:

1 Compound does not accumulate in fish. [Peer Reviewed] *DHHS/NTP; Toxicol and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) Report #309 NIH Pub #86-2565 p.19 (1986)*

2 Forty-eight hour fish bioconcentration studies with C14-labeled decabromodiphenyl ether revealed no measurable bioconcentration in fish filets(1). The measured BCF was 0.3. These results are in marked disagreement with estimates of BCF based on two regression equations using related chemicals in their basis sets. Based on the water solubility of decabromodiphenyl ether, 20-30 ppb(1), and its log octanol/water partition coefficient, 5.24(1), these equations predict BCFs of 4900 and 2500(2,SRC). The discrepancy between the observed and predicted BCF may be explained by the loss of membrane permeability that has been suggested for large hydrophobic molecules(3). [Peer Reviewed] *(1) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (2) Lyman WJ et al Handbook of Chemical Property Estimation Methods NY: McGraw-Hill Chapt 5 (1982) (3) Opperhuizen A et al: Chemosphere 14: 1871-96 (1985)*

Soil Adsorption/Mobility:

The Koc value for decabromodiphenyl ether estimated from its water solubility, 20-30 ppb(1) using a recommended regression equation is 33,000(2,SRC). Therefore decabromodiphenyl ether should be immobile in soil(3). [Peer Reviewed] *(1) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods NY: McGraw-Hill Chapt 4 (1982) (3) Swann RL et al; Res Rev 85: 17-28 (1983)*

Volatilization from Soil/Water:

The estimated Henry's Law constant for decabromodiphenyl ether is 4.45X10-8 atm-cu m/mol(1,SRC). Using this Henry's Law constant, one can predict that decabromodiphenyl ether will not volatilize from water(1). In view of its low vapor pressure, <1 mm Hg at 250 degC(2), high absorption to soil, and low Henry's Law constant, decabromodiphenyl ether would not be expected to volatilize from soil(3). [Peer Reviewed] *(1) Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991) (2) Norris JM et al; Appl Polymer Sympos 22: 195-219 (1973) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods NY: McGraw-Hill Chapt 15 & 16 (1982)*

ENVIRONMENTAL CONCENTRATIONS

Sediment/Soil Concentrations:

Decabromodiphenyl ether was found in river sediment from the Neya and Second Neya River in Osaka, Japan, 33-375 ppb (dry weight) and one of seven estuary sediments from different rivers in Japan, 20 ppb (dry weight)(1). It was not detected (detection limit 5 ppb) in two samples of marine sediment from Osaka Bay, Japan(1). Detected in soil and sediment samples near the bromine industry in Magnolia and ElDorado, AK(2). [Peer Reviewed] *(1) Watanabe I et al; Chemosphere 16: 2389-96 (1987) (2) DeCarlo VJ; Ann NY Acad Sci 320: 678-81 (1979)*

2389-96 (1987) (2) DeCarlo VJ; Ann NY Acad Sci 320: 678-81 (1979)

Atmospheric Concentrations:

SOURCE DOMINATED: Twenty-four hour samples of ambient air on the plant property of two industrial bromine extraction and bromo-organic synthesis facilities had concentrations of decabromodiphenyl ether ranging from ND to 72 ng/cu m and <13 to 25,000 ng/cu m for the two facilities(1). Detected in air particulate matter collected near the bromine industry in Magnolia and El Dorado, AK(2). [Peer Reviewed] *(1) Zweidinger RA et al; ACS Symp Ser 94: 217-31 (1979) (2) DeCarlo VJ; Ann NY Acad Sci 320: 678-81 (1979)*

Fish/Seafood Concentrations:

Found in 1 of 3 mussels from Osaka Bay, Japan at 1.4 ppb, wet weight, but not found (<0.5 ppb wet weight) in 14 other samples of fish and shellfish from Osaka Bay and other locations in Japan(1). [Peer Reviewed] *(1) Watanabe I et al; Chemosphere 16: 2389-96 (1987)*

Other Environmental Concentrations:

Detected in sludge samples from polybrominated biphenyl facilities in New Jersey(1). [Peer Reviewed] *(1) DeCarlo VJ; Ann NY Acad Sci 320: 678-81 (1979)*

HUMAN EXPOSURE

Probable Routes of Human Exposure:

1 Exposure to decabromodiphenyl ether is primarily occupational. Workers may be exposed by inhalation and dermal contact. (SRC) [Peer Reviewed]

2 NIOSH (NOES Survey 1981-1983) has statistically estimated that 544 workers are exposed to decabromodiphenyl ether in the USA(3). Industrial hygiene survey wipe samples (1977-1978) in a decabromodiphenyl ether plant in Sayreville, NJ contained 3.6 mg/sq cm and 5.9 mg/sq cm in the reactor and distillation areas, respectively(1). Personal samples in the mill area were 0.08-0.21 mg/cu m as 8-hr TWA(1). Following a spill, personal airborne levels were 1.3-1.9 mg/cu m(1). [Peer Reviewed] *(1) IARC; IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans 48: 73-84 (1989) (3) NIOSH; National Occupational Exposure Survey (NOES) (1989)*

Body Burdens:

Five composite samples of the Fiscal year 1987 National Human Adipose Tissue Survey were analyzed for decabromodiphenyl ether(1). Two samples contained 0.4 and 0.7 ppb of decabromodiphenyl ether in the lipid and one contained trace levels. Detected at concns up to 5 ppb in human hair in communities near the bromine industry in Magnolia and El Dorado, AK(2). Found in human adipose tissue obtained from a hospital in Osaka, Japan(3). [Peer Reviewed] *(1) Cramer PH et al; Mass spectral confirmation of chlorinated and brominated diphenylethers in human adipose tissues. Washington DC: USEPA-560/5-90-012 (2) DeCarlo VJ; Ann NY Acad Sci 320: 678-81 (1979) (3) IARC; IARC Monographs on the Evaluation of the*

8.0 EXPOSURE STANDARDS AND REGULATIONS

OTHER STANDARDS AND REGULATIONS

Clean Water Act Requirements:

Toxic pollutant designated pursuant to section 307(a)(1) of the Clean Water Act and is subject to effluent limitations. /Haloethers/ *40 CFR 401.15 (7/1/90)*

TSCA Requirements:

This notice announces the receipt of test data for submitted decabromodiphenyl oxide pursuant to a final test rule under the Toxic Substances Control Act (TSCA). ... The submission describes the "determination of polybrominated-p-dioxins and polybrominated dibenzofurans by high resolution gas chromatography/medium high resolution mass spectrometry. [QC Reviewed] *59 FR 27547 (5/27/94)*

9.0 MONITORING AND ANALYSIS METHODS

Analytical Laboratory Methods:

Four lots of study material were identified as decabromodiphenyl oxide by infrared and ultraviolet/visible spectroscopy. The purity of all lots of study material was determined by elemental analysis, thin layer chromatography, and high performance liquid chromatography. [Peer Reviewed] *DHHS/NTP; Toxicol and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Ratsand B6C3F1 Mice (Feed Studies) Report #309 NIH Pub #86-2565 p.22 (1986)*

10.0 ADDITIONAL REFERENCES

Special Reports:

DHHS/NTP; Toxicology & Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies) Technical Report Series No. 309 (1986) NIH Publication No. 86-2565

Decabromodiphenyl ether (DBDPE)

IRIS - Integrated Risk Information System

0035 Decabromodiphenyl ether (DBDPE); CASRN 1163-19-5 (10/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR DBDPE

File On-Line 01/31/87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/95
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	01/01/90

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5
Last Revised -- 02/01/95
The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
No adverse effects observed Rat Chronic Oral Bioassay Kociba et al., 1975	NOEL: 1.0 mg/kg/day LOAEL: none	100	1	1E-2 mg/kg/day

Liver enlargement	NOEL: 8 mg/kg/day
Rat Subchronic Oral Bioassay	LOAEL: 80 mg/kg/day
Norris et al., 1973, 1975	

*Conversion Factors: none

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Kociba, R.J., L.O. Frauson, C.G. Humiston, et al. 1975. Results of a two- year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. J. Combust. Toxicol. 2: 267-285.

Norris, J.M., J.W. Ehrmantraut, C.L. Gibbons, et al. 1973. Toxicological and environmental factors involved in the selection of decadibromophenyl oxide as a fire retardant chemical. Appl. Polym. Symp. 22: 195-219.

Norris, J.M., R.J. Kociba, B.A. Schwetz, et al. 1975. Toxicology of octabromobiphenyl and decabromodiphenyl oxide. Environ. Health Perspect. 11: 153-161.

Kociba et al. (1975) treated Sprague-Dawley (Spartan) rats (25/sex/dose) with daily doses (in the diet) of 0.0, 0.01, 0.1, and 1.0 mg decabromodiphenyl ether (oxide)/kg bw for 2 years. Parameters examined were hematology, clinical chemistry, food consumption, organ weight, body weight, and incidence of histopathologic lesions. No significant differences between treatment and control groups were found. The NOEL for this study was 1.0 mg/kg/day. Norris et al. (1973, 1975) reported on earlier stages of the same study.

Supporting data are reported by Norris et al. (1973, 1975) in a 30-day oral study, in which male rats were administered decabromodiphenyl ether (DBDPE) at dietary concentrations of 0, 0.01, 0.1, or 1.0%. These concentrations correspond to doses of 0, 8, 80, or 800 mg/kg/day. A NOEL for enlarged livers of 8 mg/kg/day was established. This short-term-to-subchronic NOEL is close to the chronic NOEL of 1.0 mg/kg/day when adjusted by a factor of 10 to account for the uncertainty in extrapolating subchronic dose to chronic dose.

The chemical analysis of the DBDPE used in these studies was reported as follows (Kociba et al., 1975): DBDPE, 77.4%; nonabromodiphenyl ether (NBDPE), 21.8%; and octabromodiphenyl ether (OBDPE), 0.8%. The presence of OBDPE in this mixture has an uncertain effect on the toxicity of DBDPE, since information on the mechanism of toxicity of DBDPE is lacking. An RfD of 0.00062 mg/kg/day has been proposed for OBDPE based on its liver enzyme reducing capabilities (U.S. EPA, 1984). No RfD has been proposed for NBDPE because of lack of data.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The 100-fold factor reflects both the expected intra- and interspecies variability to the toxicity of this chemical in lieu of specific data.

MF -- None

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Absorption and subsequent toxicity of this compound may be highly dependent on the relative proportions of water and oils in the diet because of the chemical's physical properties (low aqueous solubility and varied crystal size).

I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Medium

Study -- Medium
Data Base -- Low
RfD -- Low
The principal study was well-conducted with a sufficient number of animals and toxicity parameters, but lacked an adequate dose range. The supporting in- house study, which established the LOAEL, was very short in duration. Since no other data pertaining to the chronic toxicity of DBDPE were found in the available literature, confidence in the data base is rated low. Low confidence in the RfD follows.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1984
The ADI in the 1984 Health and Environmental Effects Profile has received an Agency review with the help of two external scientists.

Other EPA Documentation -- None
Agency Work Group Review -- 10/09/85
Verification Date -- 10/09/85

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5
Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5
Last Revised -- 01/01/90
Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- Based on no human data and limited evidence of carcinogenicity in animals; namely, significantly increased incidences of neoplastic liver nodules in male and female rats and increased incidences of hepatocellular adenomas or carcinomas (combined) in male mice.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Limited. Groups of 50 male and 50 female F344-N rats and 50 male and 50 female B6C3F1 mice were given diets containing 0, 25,000, or 50,000 ppm commercial-grade compound (94-97% pure) for 103 weeks. Average daily dosages estimated from food consumption data were 1120 and 2240 mg/kg for low- and high-dose male rats; 1200 and 2550 mg/kg for low- and high-dose female rats; 3200 and 6650 mg/kg for low- and high-dose male mice; and 3760 and 7780 mg/kg for low- and high-dose female mice (NTP, 1985). Rats and mice were maintained without treatment for an additional 1 and 0-1 weeks, respectively, and then sacrificed.

Survival of the low-dose male rats was significantly lower than controls after week 102; however, since no reduced survival was observed in any other group and the decrease was observed so late in the study, the authors concluded that the effect on survival may not have been compound related. The incidences of liver neoplastic nodules in low- and high-dose male rats (7/50 and 15/49, respectively) and high-dose female rats (9/50) were significantly greater than in controls (1/50 in both males and females). Incidences of hepatocellular carcinoma alone (1/50, control males; 1/50, low- dose males; 1/49, high-dose males; 0/50, control females; 2/49, low-dose females; 0/50, high-dose females) were not significantly increased in the treated groups compared to controls. A dose-related trend for mononuclear cell leukemia was observed in treated male rats; however, because of the high incidence in control animals, this was not considered to be biologically significant.

No evidence of carcinogenicity was observed in female mice, although there was some indication that cancer incidence had increased in male mice. Hepatocellular adenomas or carcinomas (combined) occurred at a significantly increased incidence in low-dose male mice (control, 8/50; low dose, 22/50; high dose, 18/50) after correction for survival. The incidence of hepatocellular carcinomas alone was not significant in either the low- or high-dose male mice. Treatment-related effects on body weight or clinical signs of toxicity or survival were not observed in any dose group. A high mortality of control male mice during the first year of the study, presumably due to fighting weakens the evidence of carcinogenicity in male mice. Thyroid gland follicular cell adenomas or carcinomas (combined) also occurred in treated male mice, but the increases were not significant (control, 0/50; low dose, 4/50; high dose, 3/50). The incidence of follicular cell hyperplasia in male mice was significantly increased.

Group of 25 male and 25 female Sprague-Dawley rats were given decabromodiphenyl oxide in the diet at dose levels of 0, 0.01, 0.1, or 1.0 mg/kg/day for 2 years (Kociba et al., 1975; Norris et al., 1973, 1975). The sample used contained 77.4% decabromodiphenyl oxide, 21.8% nonabromodiphenyl oxide and 0.8% octabromodiphenyl oxide. Histological examinations conducted on rats that died during the study and on those sacrificed at termination (day 702 for males and day 735 for females) did not reveal any treatment-related neoplastic effects as indicated by numbers of rats that developed tumors, total numbers of tumors, specific types of tumors or combined male and female data.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The commercial-grade compound used in the NTP (1985) carcinogenicity study was not mutagenic in Salmonella typhimurium strains TA1535, TA1537, TA98, or TA100 with or without rat or hamster liver enzyme preparations (NTP, 1985). Doses ranged from 100 to 10,000 /g/plate. Mutagenicity was not detected in the mouse lymphoma L5178Y/TK+/-assay with or without rat liver enzyme preparations at doses of 7 to 10 /g/mL (NTP, 1985). Sister chromatid exchanges or chromosomal aberrations were not induced in Chinese hamster ovary cells treated in

chromatid exchanges or chromosomal aberrations were not induced in Chinese hamster ovary cells treated in vivo at doses of 50 to 500 /g/mL with or without rat liver enzyme preparations (NTP, 1985).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987
The 1987 Health and Environmental Effects Profile for Decabromodiphenyl Oxide has received Agency review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 05/04/89
Verification Date -- 05/04/89

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5
Last Revised -- 12/01/89

VI.A. ORAL RfD REFERENCES

Kociba, R.J., L.O. Frauson, C.G. Humiston, et al. 1975. Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. J. Combust. Toxicol. 2: 267-285.

Norris, J.M., J.W. Ehrmantraut, C.L. Gibbons, et al. 1973. Toxicological and environmental factors involved in the selection of decadibromophenyl oxide as a fire retardant chemical. Appl. Polym. Symp. 22: 195-219.

Norris, J.M., R.J. Kociba, B.A. Schwetz, et al. 1975. Toxicology of octabromobiphenyl and decabromodiphenyl oxide. Environ. Health Perspect. 11: 153-161.

U.S. EPA. 1984. Health and Environmental Effects Profile for Brominated Biphenyl Ethers. Prepared by the Office

U.S. EPA. 1984. Health and Environmental Effects Profile for Brominated Biphenyl Ethers. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

VI.B. INHALATION RfC REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Kociba, R.J., L.O. Frauson, C.G. Huniston, et al. 1975. Results of a two- year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. J. Combust. Toxicol. 2(4): 267-285.

Norris, J.M., J.W. Ehrmantraut, C.L. Gibbons, et al. 1973. Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical. App. Polym. Symp. 22: 195-219.

Norris, J.M. R.J. Kociba, B.A. Schwetz, et al. 1975. Toxicology of octabromodiphenyl and decabromodiphenyl oxide. Environ. Health Perspect. 11: 153-161.

NTP (National Toxicology Program). 1985. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide in F344/N Rats and B6C3F1 Mice (Feed Studies). NTP-TR-309, NIH-85-2565.

U.S. EPA. 1987. Health and Environmental Effects Profile for Decabromodiphenyl Oxide. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office Cincinnati, OH, for the Office of Solid Waste, Washington, DC.

VII. REVISION HISTORY

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5

Date	Section	Description
03/01/88	I.A.4.	Text clarified
03/01/88	I.A.6.	Verification date changed
08/01/89	II.	Carcinogen assessment now under review
08/01/89	VI.	Bibliography on-line
12/01/89	I.A.2.	Corrected Norris et al. 1975 citation
12/01/89	II.	Carcinogen assessment on-line
12/01/89	VI.C.	Carcinogen references added
01/01/90	II.A.3.	Doses corrected
01/01/92	I.A.7.	Secondary contact changed
01/01/92	IV.	Regulatory Action section on-line
02/01/95	I.A.7.	Secondary contact's name changed

SYNONYMS

Substance Name -- Decabromodiphenyl ether (DBDPE)
CASRN -- 1163-19-5
Last Revised -- 01/31/87

1163-19-5
BENZENE, 1,1'-OXYBIS(2,3,4,5,6-PENTABROMO-
BERKFLAM B 10E
BR 55N
BROMKAL 83-10DE
BROMKAL 82-ODE
DBDPE
DBDPO
DE 83R
DECABROMOBIPHENYL ETHER
DECABROMOBIPHENYL OXIDE
Decabromodiphenyl Ether
DECABROMODIPHENYL OXIDE
DECABROMOPHENYL ETHER
ETHER, BIS(PENTABROMOPHENYL)
ETHER, DECABROMODIPHENYL
FR 300
FR 300BA
FRP 53
NCI-C55287
PENTABROMOPHENYL ETHER
SAYTEX 102
SAYTEX 102E
TARDEX 100

DECABROMODIPHENYL ETHER

Common Name: **DECABROMODIPHENYL ETHER**

CAS Number: 1163-19-5
DOT Number: None

RTK Substance number: 0598
Date: March 1992

HAZARD SUMMARY

- **Decabromodiphenyl Ether** can affect you when breathed in and may be absorbed through the skin.
- **Decabromodiphenyl Ether** can cause eye and skin irritation.
- This chemical can accumulate in the body, and may cause damage to the liver and kidneys. Thyroid enlargement (goiter) may also occur.

IDENTIFICATION

Decabromodiphenyl Ether is a white to off-white powder. It is used as a fire retardant for manmade fibers, and in thermoplastics.

REASON FOR CITATION

- **Decabromodiphenyl Ether** is on the Hazardous Substance List because it is cited by DEPE and EPA.

HOW TO DETERMINE IF YOU ARE BEING EXPOSED

- Exposure to hazardous substances should be routinely evaluated. This may include collecting personal and area air samples. You can obtain copies of sampling results from your employer. You have a legal right to this information under OSHA 1910.20.
- If you think you are experiencing any work-related health problems, see a doctor trained to recognize occupational diseases. Take this Fact Sheet with you.

WORKPLACE EXPOSURE LIMITS

No occupational exposure limits have been established for **Decabromodiphenyl Ether**. This does not mean that this substance is not harmful. Safe work practices should always be followed.

It should be recognized that **Decabromodiphenyl Ether** can be absorbed through your skin, thereby increasing your exposure.

your exposure.

WAYS OF REDUCING EXPOSURE

- Where possible, enclose operations and use local exhaust ventilation at the site of chemical release. If local exhaust ventilation or enclosure is not used, respirators should be worn.
- Wear protective work clothing.
- Wash thoroughly immediately after exposure to **Decabromodiphenyl Ether** and at the end of the workshift.
- Post hazard and warning information in the work area. In addition, as part of an ongoing education and training effort, communicate all information on the health and safety hazards of **Decabromodiphenyl Ether** to potentially exposed workers.

This Fact Sheet is a summary source of information of all potential and most severe health hazards that may result from exposure. Duration of exposure, concentration of the substance and other factors will affect your susceptibility to any of the potential effects described below.

HEALTH HAZARD INFORMATION

Acute Health Effects

The following acute (short-term) health effects may occur immediately or shortly after exposure to **Decabromodiphenyl Ether**:

- Prolonged or repeated skin contact with **Decabromodiphenyl Ether** can cause irritation or redness.
- Eye contact can cause irritation.

Chronic Health Effects

The following chronic (long-term) health effects can occur at some time after exposure to **Decabromodiphenyl Ether** and can last for months or years:

Cancer Hazard

- According to the information presently available to the New Jersey Department of Health, **Decabromodiphenyl Ether** has been tested and has not been shown to cause cancer in animals.

Reproductive Hazard

- **Decabromodiphenyl Ether** has not been shown to cause reproductive damage but it should be handled with caution since some closely related compounds (called POLYBROMINATED BIPHENYLS or PBB's) have been shown to damage the developing fetus.

Other Long-Term Effects

- This chemical can accumulate in the body with repeated exposure and may cause enlargement of

- This chemical can accumulate in the body with repeated exposure and may cause enlargement of the thyroid gland (goiter), and damage to the kidneys and liver.

MEDICAL

Medical Testing

If symptoms develop or overexposure is suspected, the following may be useful:

- Liver and kidney function tests.
- Exam of the thyroid.

Any evaluation should include a careful history of past and present symptoms with an exam. Medical tests that look for damage already done are not a substitute for controlling exposure.

Request copies of your medical testing. You have a legal right to this information under OSHA 1910.20.

WORKPLACE CONTROLS AND PRACTICES

Unless a less toxic chemical can be substituted for a hazardous substance, ENGINEERING CONTROLS are the most effective way of reducing exposure. The best protection is to enclose operations and/or provide local exhaust ventilation at the site of chemical release. Isolating operations can also reduce exposure. Using respirators or protective equipment is less effective than the controls mentioned above, but is sometimes necessary.

In evaluating the controls present in your workplace, consider: (1) how hazardous the substance is, (2) how much of the substance is released into the workplace and (3) whether harmful skin or eye contact could occur. Special controls should be in place for highly toxic chemicals or when significant skin, eye, or breathing exposures are possible.

In addition, the following control is recommended:

- Where possible, automatically transfer **Decabromodiphenyl Ether** from drums or other storage containers to process containers.

Good WORK PRACTICES can help to reduce hazardous exposures. The following work practices are recommended:

- Workers whose clothing has been contaminated by **Decabromodiphenyl Ether** should change into clean clothing promptly.
- Contaminated work clothes should be laundered by individuals who have been informed of the hazards of exposure to **Decabromodiphenyl Ether**.
- Eye wash fountains should be provided in the immediate work area for emergency use.
- On skin contact with **Decabromodiphenyl Ether**, immediately wash or shower to remove the chemical. At the end of the workshift, wash any areas of the body that may have contacted **Decabromodiphenyl Ether**, whether or not known skin contact has occurred.
- Do not eat, smoke, or drink where **Decabromodiphenyl Ether** is handled, processed, or stored, since the chemical can be swallowed. Wash hands carefully before eating or smoking.
- Use a vacuum or a wet method to reduce dust during clean-up. DO NOT DRY SWEEP.

PERSONAL PROTECTIVE EQUIPMENT

WORKPLACE CONTROLS ARE BETTER THAN PERSONAL PROTECTIVE EQUIPMENT. However, for some jobs (such as outside work, confined space entry, jobs done only once in a while, or jobs done while workplace controls are being installed), personal protective equipment may be appropriate.

The following recommendations are only guidelines and may not apply to every situation.

Clothing

- Avoid skin contact with **Decabromodiphenyl Ether**. Wear protective gloves and clothing. Safety equipment suppliers/ manufacturers can provide recommendations on the most protective glove/clothing material for your operation.
- All protective clothing (suits, gloves, footwear, headgear) should be clean, available each day, and put on before work.

Eye Protection

- Wear dust-proof goggles and face shield when working with powders or dust, unless full facepiece respiratory protection is worn.

Respiratory Protection

IMPROPER USE OF RESPIRATORS IS DANGEROUS. Such equipment should only be used if the employer has a written program that takes into account workplace conditions, requirements for worker training, respirator fit testing and medical exams, as described in OSHA 1910.134.

- Engineering controls must be effective to ensure that exposure to **Decabromodiphenyl Ether** does not occur.
- Where the potential exists for overexposure to **Decabromodiphenyl Ether**, use a MSHA/NIOSH approved full facepiece respirator with an organic vapor cartridge/ canister with high efficiency particulate prefilters. Greater protection is provided by a powered-air purifying respirator.
- Where the potential for high exposures exist, use a MSHA/NIOSH approved supplied-air respirator with a full facepiece operated in the positive pressure mode or with a full facepiece, hood, or helmet in the continuous flow mode, or use a MSHA/NIOSH approved self-contained breathing apparatus with a full facepiece operated in pressure-demand or other positive pressure mode.

QUESTIONS AND ANSWERS

Q: If I have acute health effects, will I later get chronic health effects?
A: Not always. Most chronic (long-term) effects result from repeated exposures to a chemical.

Q: Can I get long-term effects without ever having short-term effects?
A: Yes, because long-term effects can occur from repeated exposures to a chemical at levels not high enough to make you immediately sick.

Q: What are my chances of getting sick when I have been exposed to chemicals?
A: The likelihood of becoming sick from chemicals is increased as the amount of exposure increases. This is determined by the length of time and the amount of material to which someone is exposed.

Q: When are higher exposures more likely?
A: Conditions which increase risk of exposure include dust releasing operations (grinding, mixing, blasting, dumping, etc.), other physical and mechanical processes (heating, pouring, spraying, spills and evaporation from large surface areas such as open containers), and "confined space" exposures (working inside vats, reactors, boilers, small rooms, etc.).

Q: Is the risk of getting sick higher for workers than for community residents?
A: Yes. Exposures in the community, except possibly in cases of fires or spills, are usually much lower than those found in the workplace. However, people in the community may be exposed to contaminated water as well as to chemicals in the air over long periods. Because of this, and because of exposure of children or people who are already ill, community exposures may cause health problems.

The following information is available from:

New Jersey Department of Health
Occupational Health Service
Trenton, NJ 08625-0360
(609) 984-1863

Industrial Hygiene Information

Industrial hygienists are available to answer your questions regarding the control of chemical exposures using exhaust ventilation, special work practices, good housekeeping, good hygiene practices, and personal protective equipment including respirators. In addition, they can help to interpret the results of industrial hygiene survey data.

Medical Evaluation

If you think you are becoming sick because of exposure to chemicals at your workplace, you may call a Department of Health physician who can help you find the services you need.

Public Presentations

Presentations and educational programs on occupational health or the Right to Know Act can be organized for labor unions, trade associations and other groups.

Right to Know Information Resources

The Right to Know Infoline (609) 984-2202 can answer questions about the identity and potential health effects of chemicals, list of educational materials in occupational health, references used to prepare the Fact Sheets, preparation of the Right to Know survey, education and training programs, labeling requirements, and general information regarding the Right to Know Act. Violations of the law should be reported to (609) 984-5627.

DEFINITIONS

ACGIH is the American Conference of Governmental Industrial Hygienists. It recommends upper limits (called TLVs) for exposure to workplace chemicals.

TLVs) for exposure to workplace chemicals.

A carcinogen is a substance that causes cancer.

The CAS number is assigned by the Chemical Abstracts Service to identify a specific chemical.

A combustible substance is a solid, liquid or gas that will burn.

A corrosive substance is a gas, liquid or solid that causes irreversible damage to human tissue or containers.

DEPE is the New Jersey Department of Environmental Protection and Energy.

DOT is the Department of Transportation, the federal agency that regulates the transportation of chemicals.

EPA is the Environmental Protection Agency, the federal agency responsible for regulating environmental hazards.

A fetus is an unborn human or animal.

A flammable substance is a solid, liquid, vapor or gas that will ignite easily and burn rapidly.

The flash point is the temperature at which a liquid or solid gives off vapor that can form a flammable mixture with air.

HHAG is the Human Health Assessment Group of the federal EPA.

IARC is the International Agency for Research on Cancer, a scientific group that classifies chemicals according to their cancer-causing potential.

A miscible substance is a liquid or gas that will evenly dissolve in another.

mg/m^3 means milligrams of a chemical in a cubic meter of air. It is a measure of concentration (weight/volume).

MSHA is the Mine Safety and Health Administration, the federal agency that regulates mining. It also evaluates and approves respirators.

A mutagen is a substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

NCI is the National Cancer Institute, a federal agency that determines the cancer-causing potential of chemicals.

NFPA is the National Fire Protection Association. It classifies substances according to their fire and explosion hazard.

NIOSH is the National Institute for Occupational Safety and Health. It tests equipment, evaluates and approves respirators, conducts studies of workplace hazards, and proposes standards to OSHA.

NTP is the National Toxicology Program which tests chemicals and reviews evidence for cancer.

OSHA is the Occupational Safety and Health Administration, which adopts and enforces health and safety standards.

ppm means parts of a substance per million parts of air. It is a measure of concentration by volume in air.

A reactive substance is a solid, liquid or gas that releases energy under certain conditions.

A reactive substance is a solid, liquid or gas that releases energy under certain conditions.

A teratogen is a substance that causes birth defects by damaging the fetus.

TLV is the Threshold Limit Value, the workplace exposure limit recommended by ACGIH.

The vapor pressure is a measure of how readily a liquid or a solid mixes with air at its surface. A higher vapor pressure indicates a higher concentration of the substance in air and therefore increases the likelihood of breathing it in.

>>>>>>>>>EMERGENCY INFORMATION<<<<<<<<<

Common Name: **DECABROMODIPHENYL ETHER**
DOT Number: None
DOT Emergency Guide code: No Citation
CAS Number: 1163-19-5

Hazard	NJ DOH	NFPA
FLAMMABILITY	0	Not Rated
REACTIVITY	0	Not Rated
POISONOUS GASES ARE PRODUCED IN FIRE		

Hazard Rating Key: 0=minimal; 1=slight; 2=moderate; 3=serious; 4=severe

FIRE HAZARDS

- **Decabromodiphenyl Ether** may burn, but does not readily ignite.
- Use dry chemical, CO2, water spray, or foam extinguishers.
- POISONOUS GASES ARE PRODUCED IN FIRE, including Hydrogen Bromide.
- If employees are expected to fight fires, they must be trained and equipped as stated in OSHA 1910.156.

SPILLS AND EMERGENCIES

If **Decabromodiphenyl Ether** is spilled, take the following steps:

- Restrict persons not wearing protective equipment from area of spill until clean-up is complete.
- Collect powdered material in the most convenient and safe manner and deposit in sealed containers.
- Ventilate area after clean-up is complete.
- It may be necessary to contain and dispose of **Decabromodiphenyl Ether** as a HAZARDOUS WASTE. Contact your Department of Environmental Protection (DEP) or your regional office of the federal Environmental Protection Agency (EPA) for specific recommendations.

FOR LARGE SPILLS AND FIRES immediately call your fire department. You can request emergency information from the following:

from the following:

CHEMTREC: (800) 424-9300
NJDEPE HOTLINE: (609) 292-7172
Other:

HANDLING AND STORAGE

- Prior to working with **Decabromodiphenyl Ether** you should be trained on its proper handling and storage.
- **Decabromodiphenyl Ether** is not compatible with STRONG OXIDIZERS (such as CHLORINE, BROMINE and FLUORINE).
- Store in tightly closed containers in a cool, well-ventilated area.

FIRST AID

In NJ, POISON INFORMATION 1-800-962-1253
Other:

Eye Contact

- Immediately flush with large amounts of water for at least 15 minutes, occasionally lifting upper and lower lids.

Skin Contact

- Remove contaminated clothing. Wash contaminated skin with soap and water.

Breathing

- Remove the person from exposure.

PHYSICAL DATA

Vapor Pressure: 1 mm Hg at 70 °F (21 °C)
Water Solubility: Slightly soluble

OTHER COMMONLY USED NAMES

Chemical Name:
Benzene, 1,1'-Oxybis 2,3,4,5,6-Pentabromo-

Other Names:

Other Names:
bis-(Pentabromophenyl Ether); Decabromodiphenyl Oxide; DBDPO; Saytex 102; Tardex 100

NEW JERSEY DEPARTMENT OF HEALTH
Right to Know Program
CN 368, Trenton, NJ 08625-0368
(609) 984-2202

Melamine

RTECS - Registry of Toxic Effects of Chemical Substances

1.0 SUBSTANCE IDENTIFICATION

RTECS Number: OS0700000
Chemical Name: Melamine
CAS Number: 108-78-1
Molecular Formula: C3-H6-N6
Molecular Weight: 126.15
Wiswesser Notation: T6N CN ENJ BZ DZ FZ
Substance Investigated as: Tumorigen, Mutagen, Primary Irritant, Reproductive Effector
Last Revision Date: 1997

2.0 SYNONYM(S)/TRADENAME(S)

1 1,3,5-Triazine-2,4,6-triamine
2 2,4,6-Triamino-1,3,5-triazine
3 2,4,6-Triamino-s-triazine
4 Aero
5 Cyanuramide
6 Cyanuric triamide
7 Cyanurotriamide
8 Cyanurotriamine
9 DG 002 (amine)
10 Hicophor PR
11 Isomelamine
12 NCI-C50715
13 Pluragard
14 Pluragard C 133
15 s-Triazine, 2,4,6-triamino-
16 Teoham
17 Theoharn
18 Virset 656-4

3.0 HEALTH HAZARD DATA

ACUTE TOXICITY

LDLO/LCLO - LOWEST PUBLISHED LETHAL DOSE/CONC

Rat

LDLo - ROUTE: Intraperitoneal; **DOSE:** 3200 mg/kg *CODEN: 38MKAJ Bibliographic Data: "Patty's Industrial Hygiene and Toxicology," 3rd rev. ed., Clayton, G.D., and F.E. Clayton, eds., New York, John Wiley & Sons, Inc., 1978-82. Vol. 3 originally pub. in 1979; pub. as 2nd rev. ed. in 1985. CODEN Reference: 2A:2769,1981*
TOXIC EFFECTS:
Sense Organs and Special Senses (Nose, Eye, Ear, and Taste) - Lacrimation
Behavioral - Tremor
Lung, Thorax, or Respiration - Cyanosis

Mouse

Mouse

LDLo - ROUTE: Intraperitoneal; **DOSE:** 800 mg/kg *CODEN: 38MKAJ Bibliographic Data: "Patty's Industrial Hygiene and Toxicology," 3rd rev. ed., Clayton, G.D., and F.E. Clayton, eds., New York, John Wiley & Sons, Inc., 1978-82. Vol. 3 originally pub. in 1979; pub. as 2nd rev. ed. in 1985. CODEN Reference: 2A:2769,1981*
TOXIC EFFECTS:
Sense Organs and Special Senses (Nose, Eye, Ear, and Taste) - Lacrimation
Behavioral - Tremor
Lung, Thorax, or Respiration - Cyanosis

LD50/LC50 - LETHAL DOSE/CONC 50% KILL

Rat

LC50 - ROUTE: Inhalation; **DOSE:** 3248 mg/m3 *CODEN: GISAAA Bibliographic Data: Gigiena i Sanitariya. For English translation, see HYSAAV. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1- 1936- CODEN Reference: 58(2):14,1993*
LD50 - ROUTE: Oral; **DOSE:** 3161 mg/kg *CODEN: TXAPA9 Bibliographic Data: Toxicology and Applied Pharmacology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1- 1959- CODEN Reference: 72:292,1984*
LD50 - ROUTE: Unreported; **DOSE:** 6 gm/kg *CODEN: GISAAA Bibliographic Data: Gigiena i Sanitariya. For English translation, see HYSAAV. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1- 1936- CODEN Reference: 58(2):14,1993*

Mouse

LD50 - ROUTE: Oral; **DOSE:** 3296 mg/kg *CODEN: TXAPA9 Bibliographic Data: Toxicology and Applied Pharmacology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1- 1959- CODEN Reference: 72:292,1984*
LD50 - ROUTE: Unreported; **DOSE:** 1 gm/kg *CODEN: GISAAA Bibliographic Data: Gigiena i Sanitariya. For English translation, see HYSAAV. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1- 1936- CODEN Reference: 58(2):14,1993*

Rabbit

LD50 - ROUTE: Skin; **DOSE:** >1 gm/kg *CODEN: ATDAEI Bibliographic Data: Acute Toxicity Data. Journal of the American College of Toxicology, Part B. (Mary Ann Liebert, Inc., 1651 Third Ave., New York, NY 10128) V.1- 1990- CODEN Reference: 1:110,1990*

IRRITATION

EYE - STANDARD DRAIZE TEST

Rabbit

ROUTE: Eyes; **DOSE:** 500 mg/24H; **REACTION:** Mild *CODEN: 28ZPAK Bibliographic Data: "Sbornik Vysledku Toxixologickeho Vysetreni Latek A Pripravku," Marhold, J.V., Institut Pro Vychovu Vedoucicn Pracovniku Chemickeho Prumyclu Praha, Czechoslovakia, 1972 CODEN Reference: -,153,1972*

REPRODUCTIVE EFFECTS

Rat

ROUTE: Inhalation; **DOSE:** 500 ug/m3; **DURATION:** male 17W prior to mating *CODEN: GISAAA Bibliographic Data: Gigiena i Sanitariya. For English translation, see HYSAAV. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1- 1936- CODEN Reference: 58(2):14,1993*
TOXIC EFFECTS:
Paternal Effects - Spermatogenesis (including genetic material, sperm morphology, motility, and count)
Effects on Fertility - Pre-implantation mortality (e.g., reduction in number of implants per

female; total number of implants per corpora lutea)
Effects on Embryo or Fetus - Fetal death

GENETIC EFFECTS

MICRONUCLEUS TEST

Mouse

ROUTE: oral; **DOSE:** 1 gm/kg *CODEN: ENMUDM Bibliographic Data: Environmental Mutagenesis. (New York, NY) V.1-9, 1979-87. For publisher information, see EMMUEG CODEN Reference: 4:342,1982*

PHAGE INHIBITION

Bacteria - E Coli

DOSE: 78 ug/well *CODEN: MUREAV Bibliographic Data: Mutation Research. (Elsevier Science Pub. B.V., POB 211, 1000 AE Amsterdam, Netherlands) V.1- 1964- CODEN Reference: 260:349,1991*

TUMORIGENIC EFFECTS

Rat

ROUTE: Oral; **DOSE:** 162 gm/kg/2Y continuous *CODEN: TXAPA9 Bibliographic Data: Toxicology and Applied Pharmacology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1- 1959- CODEN Reference: 72:292,1984*
TOXIC EFFECTS:
Tumorigenic - Equivocal tumorigenic agent by RTECS criteria
Kidney, Ureter, and Bladder - Tumors
ROUTE: Oral; **DOSE:** 195 gm/kg/2Y continuous *CODEN: TXAPA9 Bibliographic Data: Toxicology and Applied Pharmacology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1- 1959- CODEN Reference: 72:292,1984*
TOXIC EFFECTS:
Tumorigenic - Carcinogenic by RTECS criteria
Kidney, Ureter, and Bladder - Tumors
ROUTE: Oral; **DOSE:** 197 gm/kg/2Y continuous *CODEN: NTPTR* Bibliographic Data: National Toxicology Program Technical Report Series. (Research Triangle Park, NC 27709) No.206- CODEN Reference: NTP-TR-245,1983*
TOXIC EFFECTS:
Tumorigenic - Carcinogenic by RTECS criteria
Kidney, Ureter, and Bladder - Tumors

OTHER MULTIPLE DOSE TOXICITY DATA

Rat

ROUTE: Inhalation; **DOSE:** 58 ug/m3/17W intermittent *CODEN: GISAAA Bibliographic Data: Gigiena i Sanitariya. For English translation, see HYSAAV. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1- 1936- CODEN Reference: 58(2):14,1993*
TOXIC EFFECTS:
Liver - Other changes
Blood - Changes in serum composition (e.g., TP, bilirubin, cholesterol)
Biochemical - Transaminases
ROUTE: Oral; **DOSE:** 32760 mg/kg/13W continuous *CODEN: NTPTR* Bibliographic Data: National Toxicology Program Technical Report Series. (Research Triangle Park,*

NC 27709) No.206- CODEN Reference: NTP-TR-245,1983
TOXIC EFFECTS:
Kidney, Ureter, and Bladder - Other changes
Others - Death

Mouse

ROUTE: Oral; **DOSE:** 93600 mg/kg/13W continuous *CODEN: NTPTR* Bibliographic Data: National Toxicology Program Technical Report Series. (Research Triangle Park, NC 27709) No.206- CODEN Reference: NTP-TR-245,1983*
TOXIC EFFECTS:
Kidney, Ureter, and Bladder - Other changes

5.0 NIOSH DOCUMENTS

1 National Occupational Exposure Survey 1983: Hazard Code 81887; Number of Industries 27; Total Number of Facilities 2132; Number of Occupations 44; Total Number of Employees 43104; Total Number of Female Employees 6239
2 National Occupational Hazard Survey 1974: Hazard Code 81887; Number of Industries 32; Total Number of Facilities 1043; Number of Occupations 49; Total Number of Employees 31892

6.0 REVIEWS

1 IARC Cancer Review:Animal Inadequate Evidence *CODEN: IMEMDT Bibliographic Data: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. (WHO Publications Centre USA, 49 Sheridan Ave., Albany, NY 12210) V.1- 1972- CODEN Reference: 39:333,1986*
2 IARC Cancer Review:Group 3 *CODEN: IMSUDL Bibliographic Data: IARC Monographs, Supplement. (WHO Publications Centre USA, 49 Sheridan Ave., Albany, NY 12210) No.1- 1979- CODEN Reference: 7:56,1987*
3 IARC Cancer Review:Human No Adequate Data *CODEN: IMEMDT Bibliographic Data: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. (WHO Publications Centre USA, 49 Sheridan Ave., Albany, NY 12210) V.1- 1972- CODEN Reference: 39:333,1986*
4 TOXICOLOGY REVIEW *CODEN: ECIET* Bibliographic Data: European Chemical Industry Ecology and Toxicology Centre. Joint Assessment of Commodity Chemicals. (Avenue Louise 250, B-63, B-1050 Brussels, Belgium) CODEN Reference: (1),-,1983*

7.0 STATUS IN U.S.

1 EPA GENETOX PROGRAM 1988, Inconclusive: D melanogaster Sex-linked lethal
2 EPA TSCA Section 8(b) CHEMICAL INVENTORY
3 EPA TSCA Section 8(d) unpublished health/safety studies
4 EPA TSCA TEST SUBMISSION (TSCATS) DATA BASE, SEPTEMBER 1997
5 NTP Carcinogenesis Bioassay (feed);clear evidence:rat NTPTR* NTP-TR-245,83
6 NTP Carcinogenesis Bioassay (feed);no evidence:mouse NTPTR* NTP-TR-245,83